

SEWILAM'S CLINICAL MEDICINE



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GENERAL HISTORY

❖ General History is composed of 5 main parts :

A. Personal History

- Name** (الاسم ثلاثي) : اسم حضرتك اي
 - To be familiar with the patient & to gain confidence.
- Age** : ؟ سنة ؟
 - Young age : congenital
 - Old age : atherosclerosis
- Sex**:
 - Females : SLE , Myxoedema
 - Males : COPD , Coronary heart disease
- Occupation** : ؟ بتشتغل اي ؟ ولو بطل شغل ، بطلت ليه ؟
 - Glass workers : silicosis
 - Dusty workers : pneumoconiosis
- Marital status**: ؟ متزوج من امتي ؟ عندك اولاد ؟ كام ولد ؟ اصغرهم عنده كام سنة ؟
 - Single, married, divorced, widow (widower)
 - Duration , Number of children & age of the youngest
- Menstrual history** if female patient :
 - Menarche or menopause : اول مرة جتلك الدورة امتي ، لو الدورة قاطعة : اسأل عن وسائل منع الحمل بس
 - Last menstrual period : آخر مرة جتلك الدورة امتي
 - Regular or not : الدور منتظمة ولا لا
 - Time of period & length : بتيجي كل اديه ؟ وبتكون معاكاي كام يوم ؟
 - Amount : How many pads she changes per day (بتغيري كام فوطه)
 - Lactation : Breast feeding or not : رضعتي ولادك طبعي ؟
 - Contraception : which method & for how long : استخدمتي وسائل لمنع الحمل وايه ولمدة ادي ؟

Example : her menarche was at age of 12 years. It is regular every 28 day , lasting for 5 days of average amount. she was breastfeeding , with no history of usage of contraception
- Residence & address** : ؟ ساكن فين ؟ مولد وعائش طول عمرك هناك ؟
 - Born in & lives in ...
 - Endemic areas for parasitic diseases.
- Habits of medical importance**:
 - Smoking : ؟ بتدخن ؟ كام سيجارة في اليوم ؟ من كام سنة ؟ بطلت ؟ امتي ؟ وليه ؟
 - Type, number of cigarettes/day, duration
 - Smoking index = Cigarettes/day X duration in years
 - Mild smoker < 100
 - Moderate smoker : 100 – 400
 - Heavy smoker > 400
 - Others: Shisha, Alcohol, cannabis (hashish), morphine, heroin, drugs.
 - بتشرب خمره أو مخدرات ؟ بطريقة منتظمة ولا في المناسبات ؟
- Handedness** : ؟ بتكتب بإيدك اليمين ولا الشمال ؟
 - To determine dominant hemisphere in neurological cases.
 - If dominant hemisphere is affected, aphasia occurs.
 - For more details , see neurological sheet.

❖ Personal history:

- ✓ ناس أمرا NAS OMMRHH
- ✓ Write personal History in a single paragraph.



Example of personal History:

- Male patient , Zeyad Saeed Soliman, 55 years old, doctor, married and has 2 children, the youngest is 4 years old, was born and lives in Cairo, he is cigarette smoker, he smokes 20 cigarettes/day for 20 years & he is right handed.

B. Complaint + duration: ؟ إي اللي جايك المستشفى ؟

❖ As short as possible.

❖ No medical terms , in patient own words e.g.

• Abdominal distension not ascites	• Shortness of breath not dyspnea
• Awareness of heart beat not palpitation	• Swelling of both lower limbs not edema of lower limbs

❖ E.g.: the patient is complaining of shortening of breathing of 1 week duration

C. History of present illness = HPI ؟ المرض بدأ معاك إزاي , آخر مرة كنت كويس فيها إمتي ؟

1. Use medical terms , Long

2. Special analysis : according to every sheet

3. General analysis of complaint (OCD ↑↓ SIT):

○ Onset: acute, gradual, insidious ؟ ظهر فجأة ولا بالتدريج ؟

○ Course: progressive, regressive, stationary, remission & exacerbation

○ التعب بيقل ولا بيزيد ولا بيحدث في صورة نوبات ولا زي ما هو

○ Duration ؟ التعب ده بقاله أد اي ؟

○ هل مع التعب كان فيه اي اعراض تانية ؟

○ Associated Symptoms by leading questions

○ What increases ↑ & what decreases ↓ ؟

○ Related Investigation ؟ أشعات أو تحاليل ؟

○ Related Treatment (medical or surgical) & it's effect

○ روجت مستشفى , اخدت ادوية وتأثير العلاج اي بتقل الأعراض ولا لسه زي ما هي , عملت عمليات ؟

4. Review for other systems

❖ HPI :

✓ Analysis the complaint

✓ Positive data in chronological order

✓ Negative data are mentioned as well, related then non related.

D. Past history (DODs) ; events before onset of HPI:

1. Diseases:

○ Coronary heart disease , rheumatic fever , tonsillitis

○ Tuberculosis. ؟ جالك زמן كحة ودم وبلغم ؟ كنت بتسخن بليل وتبل الملايه عرق ؟ اتحجرت في مستشفى الصدر وأخذت علاج لفترة طويلة ؟

○ DM, ؟ عندك سكر ؟ من إمتي ؟ اخدت علاج ايه , جرعة اد اي ؟ آخر تحليل سكر كان كام ؟ حصل اي مضاعفات ؟

○ Hypertension ؟ عندك ضغط ؟ من إمتي ؟ اخدت علاج ايه , جرعة اد اي ؟ آخر مرة قيس الضغط كان كام ؟ حصل اي مضاعفات ؟

→ (some schools of clinical medicine prefer to mention them in HPI; the patient known to be ... diabetic or hypertensive)

○ Hepatitis ؟ لون عينك اصفر ؟ لون البول اتغير ؟ رحت الحميات ؟

○ Bilharziasis ؟ جالك بلهارسيا قبل كده ؟ نزلت التربة قبل كده وجالك دم في البول أو البراز ؟ لو نعم كمل أسئلة

○ الحكاية ديه من إمتي ؟ اخدت علاج اي ؟ جرعة اد اي ؟ حللوك بعدها وقالوك خفيت ولا لا ؟ نزلت التربة بعد كده ؟

○ عملت عملية قبل كده ؟ عملية اي ؟ من إمتي ؟ حصل بعدها مضاعفات أو تلوث في الجرح ؟ انتفك دم ؟

2. Operations & blood transfusion: ؟

❖ N.B.:

▪ If the operation is before onset of HPI, mention it with past history.

▪ If the operation is after onset of HPI, mention it with HPI.

➤ The patient was operated upon ... years ago, for ... "name of operation" in ..., complicated by ... or not complicated

3. Drug intake: type & duration & Drug allergy. ؟ بتأخذ ادوية بصفة مستمرة ؟ عندك حساسية من اي دوا ؟

4. Similar attacks ؟ عملت حوادث قبل كده ؟ Trauma & هل جت لك الحالة ديه قبل كده ؟

E. Family history:

1. Similar condition in family ؟ حد في العائلة اشتكى بنفس الشكوى

2. Positive consanguinity ؟ الأب والأم قرابين ؟

3. Common diseases : DM , HTN , TB ؟ في حد في العائلة عنده ضغط او سكر او درن (سل) ؟

➤ In negative cases → irrelevant FH "of no clinical significance"

GENERAL EXAMINATION

I. Vital signs	II. General overview (A, B, C, D, E)	III. Systemic overview
1. Temperature	A. Appearance	1) Head & neck
2. Pulse	B. Built	2) Upper limb
3. Blood pressure	C. Consciousness / Color	3) Lower limb
4. Respiratory movements	D. Decubitus	4) Other systems except that of local examination.
	E. Facial Expression	

I. Vital signs

1. Temperature

❖ **Measurement :**

1. Sterilize the thermometer in 70% alcohol for at least 20 minutes
2. Shake the mercury down to 35 °C using a snapping wrist motion then put it in for at least 3 minutes:

○ The mouth (under the tongue with closed lip)	○ Axilla	○ Rectum (3-4 inches in the anal canal)
 Oral test of body temperature		
○ Normal oral temperature : ▪ 36.5 – 37.2 °c	○ Axillary temperature: ▪ Add 0.5 °c	○ Anal temperature (core temperature) : ▪ Subtract 0.5 °c ▪ Patient lie on one side with hip flexed ▪ Lubricate the thermometer

❖ **N.B.:**

- °F (Fahrenheit) = (°C x 9/5) + 32
- Normal diurnal variation < 1 °C : lowest in the early morning reaching peak in afternoon then decreases again, this is due to heat regulating center in the brain

❖ **What is Pulse: temperature ratio?**

- Every rise of body temp by 1 °C ↑ pulse by 10-15 beats due to the effect of temperature which causes more steep rapid diastolic depolarization of action potential of SAN.

❖ **What are the Causes of Higher pulse rate (more than 15 bpm) for pyrexia "relative tachycardia" ?**

- Carditis (rheumatic, infective, diphtheritic)

❖ **What are the Causes of Slower pulse rate for a given pyrexia (less than 10 bpm) "relative bradycardia" ?**

- Typhoid fever , Viral infection , Meningitis
- Ascending cholangitis in obstructive jaundice "Charcot's fever"

❖ **What are the indications to measure the temperature per anus = what are the conditions you cannot measure the temperature orally?**

A. Child, Convulsion, Coma	B. Dyspnea & mouth breather
C. Difficulty : painful mouth disease(ulcers) , insane, trismus of jaw, severe vomiting	

❖ **What are the grades of fever ?**

• Grade	1. Low grade fever	2. High- grade fever	3. Hyperpyrexia
• Range	○ From 37.2 to 38.5 °C	○ From 38.6 to 40.5 °C	○ ≥ 40.6 °C
• Causes	○ Malignancy ○ Infection		○ Heat stroke, Pontine Hemorrhage ○ Encephalitis, Status Epilepticus ○ Thyrotoxic crisis

- ❖ What is meant by hypothermia & what are the causes ?
- Hypothermia : $\leq 35^{\circ}\text{C}$
- Causes: (AAP, Mahmoud Sewilam)
- Old Age, damage of Anterior hypothalamus, Phenothiazine, Myxedema, Starvation & Shock.
- ❖ What are types of fever?

Types of fever

The figure displays four graphs on a grid background, each representing a different pattern of fever:

- 1. Continuous:** The temperature curve is always above the normal baseline and shows small, frequent fluctuations, remaining relatively high throughout the day.
- 2. Remittent:** The temperature curve is always above the normal baseline and shows larger daily fluctuations, but it never returns to the normal level.
- 3. Intermittent or hectic:** The temperature curve shows distinct peaks above the normal baseline, followed by periods where it returns to the normal level.
- 4. Relapsing or cyclic:** The temperature curve shows periods of high fever (above the normal baseline) that are completely interrupted by periods of normal temperature.

Type	1. Continuous	2. Remittent	3. Intermittent or hectic	4. Relapsing or cyclic
Description	Temperature is always high & daily fluctuations less than 1°C	Temperature is always high & daily fluctuations more than 1°C	Temperature falls to normal level once or more during the day	Short days of fever intercepted by short days of normal temperature.
Example	- Typhoid - Pneumonia	- Empyema - Pus under tension	Benign tertian malaria	Brucellosis, Hodgkin's disease

❖ What are the causes of fever (pyrexia) of unknown origin (FUO = PUO) ?

- Definition : an illness lasting **more than 3 weeks** with temperature **above 38.3°C** in which diagnosis has not been made **despite** a good hospital evaluation.
- Causes :

1. Infectious: • Abscesses : hepatic, splenic & perinephric • Protozoal : malaria, amoebic liver abscess & toxoplasmosis • Bacterial : SBE, typhoid, brucellosis, pneumonia & pyelonephritis • Viral : IMN, CMV, AIDS & Hepatitis • Granuloma : TB & fungal	2. Non infectious inflammation: • Collagen diseases: RF, FA, SLE, PAN • Granuloma : sarcoidosis, Crohn's disease • Tissue injury: pulmonary emboli
3. Drug fever : sulphonamides, penicillins & barbiturates	4. Psychogenic fever: factitious fever
5. Neoplastic diseases: • Blood malignancies : lymphoma & leukemia • Solid malignancies : Cancer kidney, liver, pancreas, GIT & lung.	6. Miscellaneous: • FMF, thyroiditis & hemolysis

❖ What is the difference between fever & hyperthermia?

❖ Fever	❖ Hyperthermia
❖ Pathogenesis:	
○ Raised thermostat set-point ○ Elevated levels of PGE2 in the hypothalamus is the trigger for raising thermostat set-point → stimulate vasomotor center → peripheral vasoconstriction → ↓ heat loss from the skin (cold sensation) → ↑ raise core-body temperature 1 or 2°C	○ Normal thermostat set-point ○ Due to ▪ Exogenous heat exposure (heat stroke) or ▪ Endogenous heat production (thyrotoxic crisis) or ▪ Drugs (atropine, succinyl choline, malignant neuroleptic syndrome)
❖ Treatment:	
○ Antipyretics give good response	○ Physical measures (not antipyretics)

2. Pulse

❖ Comment on the following: (RR-VV-SEP), others.

- Rate, Rhythm
- Volume, Condition of the Vessel wall
- Special character, Equality on both sides in pulse volume, Peripheral pulsations

1. Rate :

- Count it in 1 minute.
- Site: in radial artery between the radial styloid & tendon of flexor carpi radialis using middle 3 fingers with semi-pronated hand to avoid stretch of brachial artery by fascia in cubital fossa.
- Normal rate : 60-100 b/m
- Bradycardia : < 60 b/m
- Tachycardia: > 100 b/m



❖ What are the causes of :

	Bradycardia	Tachycardia
• Rate	▪ < 60 b/m	▪ > 100 b/m
• Physiological	▪ Athletes, deep sleep	▪ Emotional & mental stress
• Pathological	▪ Obstructive jaundice ▪ Hypothyroidism	▪ Hypovolemia ▪ Hyperthyroidism
• Pharmacological	▪ β -Blockers, Ca-channel blocker, digitalis	▪ Adrenaline, atropine

2. Rhythm :

- Normal : regular
- Abnormally: Irregular :
 - Tachycardia or bradycardia with variable AV block
 - AF; atrial fibrillation
 - Multiple premature beats = extrasystole

❖ How to differentiate between A.F. & extrasystole ?

	AF	Extrasystole
• Rate	▪ Rapid 120 - 150 / min. ▪ Rare slow AF ❖ Causes of slow AF: ○ Drugs : Digitalis or β-blockers ○ Lone AF ○ Associated heart block	▪ Normal
• Rhythm	▪ Marked irregularity (can't count 4 regular beats) (irregular irregularity)	▪ Occasional irregularity (can count 4 regular beats) (regular irregularity)
• Pulsus deficit	▪ > 10 / min.	▪ < 10 / min.

❖ Pulsus deficit: done in irregular pulse

- Definition : apical pulse rate (as detected by counting S1 by stethoscope) > radial pulse
- Cause : ventricular contraction opening cusps without peripheral transmission of blood

• 1st sound	▪ Variable	▪ Normal with occasional irregularity
• JVP	▪ Absent a wave	▪ Normal with occasional irregularity
• Exercise	▪ ↑ Irregularity	▪ ↓ irregularity
• ECG	▪ Absent a wave - irregular QRS	▪ Early QRS complex followed by compensatory pause

3. Volume = amplitude = pulse pressure (PP) = degree of expansion:

- Normally pulse pressure = (Systolic – Diastolic) = $120 - 80 = 40$ mmHg
 - Average range : $30 \rightarrow 60$ mmHg i.e. average volume
- Abnormally : small volume, big volume, variable volume .

❖ Causes of small volume (weak pulse) = narrow PP = low CO ❖ < 30 mmHg	❖ Causes of big volume (wide PP) = prominent arterial pulsation = causes of hyperdynamic circulation ❖ > 60 mmHg	❖ Causes of variable volume
• ↓ Filling : ○ Pericardial effusion ○ Constrictive pericarditis	• AR • Atherosclerosis, • Anemia, • AV fistulae	• Arrhythmia: ○ AF ○ Multiple extrasystoles
• ↓ Pumping: ○ Myocardial infarction ○ Heart failure ○ Shock	• Beri Beri. • Block of heart, bradycardia. • Paget disease, pregnancy, ↑BP (systemic hypertension)	• Variable partial heart block
• Obstruction: ○ AS, MS. ○ Pulmonary hypertension	• Thyrotoxicosis	• Pulsus alternans

4. Condition of the vessel wall :

- Normally: not felt
- Abnormally: felt
 - Thickened (cord like) in focal arteriosclerosis "Monckeberg sclerosis"
 - Beaded in polyarteritis nodosa
- Technique: by Rolling technique: Using 3 finger:



استخدم ثلاث اصابع , الاصبعين الطرفيين لمنع الدم والاصبع الأوسط يعمل بعمل Rolling ♥

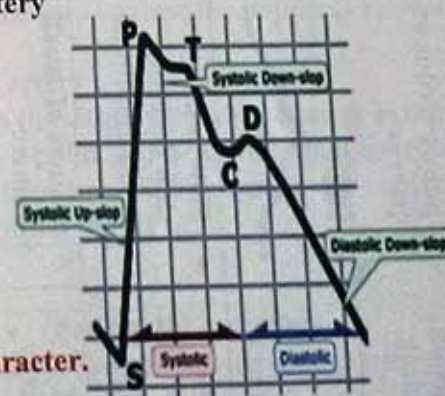
1. Place the tip of three fingers (ring, middle, index) over the radial artery
2. Press proximally using the index finger to close the radial artery
3. Press by the ring finger distally to prevent the back flow
4. Palpate the vessel wall by the middle finger

5. Special character :

- ❖ Normally:
 - **Normal pulse : gradual ascent & gradual descent i.e. no special character.**

❖ The normal pulse :

1. **S (starting point)**: starting point of arterial pulse-wave. Aortic valve opens and the blood of the left ventricle is discharged.
2. **P (percussion wave)**: wave caused from LV ejection that increase the arterial wall linearly.
3. **T (tidal wave)**: reflected wave from the small artery.
4. **C (incisura)** : end-point of systolic phase, then aortic valve is closed.
5. **D (dicrotic wave)**: reflective oscillatory wave occurred from the blood crash into aortic valve by blood pressure of aorta.



- ❖ Abnormally : see the table :

Special character	1. Water hammer pulse = collapsing pulse = bounding pulse	2. Plateau pulse = pulsus tardus et parvus	3. Pulsus Bisferiens	4. Pulsus alternans	5. Pulsus bigeminus or trigeminus = extrasystoles	6. Pulsus paradoxus
Picture						
Definition	• Sudden ascent, sudden descent, big amplitude	• Slow ascent, slow descent, small amplitude	• Pulse with 2 palpable systolic peaks	• A strong beat followed by a weak beat with equidistance	• A strong beat followed by a weak beat then a pause	• ↓ pulse volume with deep inspiration due to ↓ systolic BP more than 10 mmHg
Causes	Causes of big volume especially AR	Causes of small volume especially AS	- Double aortic lesion (AR + AS) - Idiopathic hypertrophic subaortic stenosis	Left ventricular failure	- Digitalis toxicity - Hypokalemia - Myocardial infarction	- Cardiac tamponade : pericardial effusion & constrictive pericarditis - Obstructive lung disease : COPD, status asthmaticus



❖ Why is the name "water hammer" ?	❖ How to detect water hammer pulse?
It is a popular toy in the 19th century formed of evacuated glass bottle half filled with water, when held in the hand and suddenly inverted, it delivers short hard knock.	أيدك الشمال تمسك إيد العين , واليمين تحوط ذراع المريض , وتحس خبطات قويه Best elicited by raising patient arm high (to ↓ diastole) while palpating forearm by palm.

- ❖ **What is the mechanism of pulsus alternans?**
- Alternating strong (healthy & diseased) & weak (healthy only) regular beats due to prolonged recovery time of damaged myocardium because of heterogeneity of refractoriness of the failed myocardium
- ❖ **How to detect pulsus alternans?**
- By palpating the pulse.
- By sphygmomanometer. Measuring BP by elevation of the cuff pressure above systolic BP then very gradual release of pressure till feeling the pulse, at this pressure hold the cuff inflation and count the pulse, then remove the cuff and recount the pulse, if it's doubled so pulsus alternans is diagnosed
- ❖ **How to detect pulsus paradoxus ?**
- It is misnomer because it is an exaggeration of the normal pattern and not a paradox
- In normal subjects, during inspiration, the lungs expand & accommodate more amount of blood, thus there will be a normal ↓ SBP (less than 10 mmHg) & ↓ pulse volume.
- In abnormal subjects : e.g. pericardial effusion during inspiration, there will be an exaggerated ↓ SBP (more than 10 mmHg) an exaggerated ↓ in pulse volume
- It can be detected by palpation of the arterial pulse or by detecting ↓ SBP more than 10 mmHg during inspiration using a sphygmomanometer.
- ❖ **What are the special characters of pulse associated with aortic valve disease?**

• AR: water hammer pulse	• AS: Plateau pulse	AR + AS : Pulsus Bisferiens
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6. Equality on both sides of pulse volume :



- Both radial arteries should be palpated at the same time

❖ Causes of unequal pulse volume:

- In the Lumen: thrombosis, embolism
- In the Wall:
 - Aneurysm of aortic arch
 - Dissecting aneurysm of aorta
 - Arteritis
 - Atypical coarctation of aorta involving left subclavian
- Outside the wall: cervical rib, pancost tumor

❖ Causes of unequal carotid pulsations:

- Aneurysm of aortic arch
- Dissecting aneurysm of aorta
- Atheroma of innominate artery or the common carotid

7. Peripheral pulsations on BOTH sides:

A. Head & Neck	B. Upper Limb	C. Lower Limbs
- Superficial temporal artery : - anterior & superior to the tragus - Facial artery : - In front of anterior border of masseter - Common & external carotid artery: - At the middle 1/3 of anterior border of sternomastoid, then press against carotid tubercle which is the anterior tubercle of transverse process of C6 - By thumb or tips of index & middle fingers - NEVER to feel both side at the same time. ❖ N.B. : internal carotid artery is felt in the tonsillar fossa.	- Radial artery: - Between the radial styloid & tendon of flexor carpi radialis using middle 3 fingers with semi-pronated hand to avoid stretch of brachial artery by fascia in cubital fossa - Brachial artery : - Upper part: felt by pressing along the medial border of biceps muscle - Lower part: Flex the elbow, using the thumb, felt by pressing along the medial border of biceps tendon - Axillary artery: - Put 2 thumb on the deltoid muscle & all fingers backwards to feel the pulse	- Dorsalis pedis artery : - First metatarsal space, just lateral to tendon extensor hallucis longus ❖ N.B.: - May be absent as normal variant in 15% - If dorsalis pedis pulse is felt, you must examine pulsation in posterior tibial artery as dorsalis pedis is the continuation of anterior tibial artery not the posterior. - Posterior tibial artery : - Felt midway between medial malleolus & tendo-achilhis - Popliteal artery: at popliteal fossa: - Upper part: the patient lie on his face (prone) with the knee flexed, feel the pulse against the popliteal surface of the femur - Lower part: The patient lying on the back (supine), flex the knee at right angle & use the 2 thumbs on tibial tuberosity & press all other fingers against the popliteal surface of the tibia - Femoral artery : - Put thigh in flexion with abduction & external rotation, a point just below inguinal ligament midway between ASIS & symphysis pubis (mid inguinal point)

❖ What are the causes of absent or decreased peripheral pulsation?

- Leriche's syndrome :
 - Claudication of the buttocks and thighs
 - Absent or decreased femoral pulses
 - Impotence

- Coarctation of aorta
- Extensive atherosclerosis

- Peripheral embolism
- Thromboangitis obliterans "Burger's disease"

8. others :

- Force → systolic BP : least pressure needed to occlude arterial pulsation, it's a rough estimation of SBP
- Tension → diastolic BP : least pressure needed to feel arterial pulsation, it's a rough estimation of DBP
- Radiofemoral delay: femoral artery is weaker & delayed when compared with radial pulse simultaneously in coarctation of aorta.

❖ Example of full comment on the normal pulse:

- ✗ The patient's pulse is regular, 75b/m, average volume, equal in both sides, no special character, vessel wall not felt, with intact peripheral pulsations & no radio-femoral delay

❖ Example of full comment on pulse in case of AF:

- ✗ The patient's pulse is irregular, 75b/m, variable volume, pulsus deficit > 10, equal in both sides, no special character, vessel wall not felt, with intact peripheral pulsations & no radio-femoral delay

Superficial temporal artery



Radial artery:



Dorsalis pedis artery :



Facial artery :



Brachial artery : upper part



Posterior tibial artery :



External carotid artery



Brachial artery : lower part



Popliteal artery: upper part



Water-hammer pulse



Axillary artery



Popliteal artery: lower part



Femoral artery :



Radio-femoral timing :



3. Blood Pressure

❖ Definition :

- **Systolic BP:** pressure exerted on the arterial wall by blood ejected from LV contraction.
- **Diastolic BP:** pressure exerted on the arterial wall as a result of elastic recoil of aorta.

❖ Values :

- ❖ Normal range : $\frac{\text{Systolic from 90 to 140 mmHG}}{\text{Diastolic from 60 to 90 mmHg}}$

Category	Systolic BP	Diastolic BP
• Optimal	< 120	< 80
• Normal	< 130	< 85
• High normal	130-139	85-89
Hypertension		
• Mild (stage 1)	140 - 159	90 - 99
• Moderate (Stage 2)	160 - 179	100 - 109
• Severe (Stage 3)	≥ 180	≥ 110
Isolated systolic hypertension		
• Grade I	140 - 159	< 90
• Grade II	≥ 160	< 90

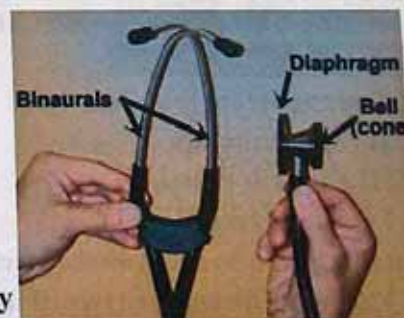
❖ Types of sphygmomanometer :

- **Mercurial :** the oldest and the most accurate
- **Aneroid :** without fluid "pneumatic"
- **Electronic:** the most recent & least accurate



❖ What are the uses of Sphygmomanometer?

1. Record BP + staging of hypertension
2. Detect pulsus alternans
3. Detect pulsus paradoxus
4. Trousseau sign : elicit carpopedal spasm in latent tetany
5. Lowenberg's cuff sign : elicit pain in DVT
6. Hess test : elicit $\uparrow$ capillary fragility in purpura
7. Walker's test : elicit ptosis in myasthenia gravis
8. Therapeutic :
 - Closed venesection : in acute HF, hypertensive encephalopathy
 - Secure hemostasis



❖ Measurement:

A. Palpatory method:	B. Auscultatory method :
• Used for systolic BP only • To avoid auscultatory gap (silent interval between systolic and diastolic pressure) • Less accurate • Method : see below.	• Used for systolic & diastolic BP • Method : see below.

❖ What is auscultatory gap & how to avoid ?

➤ Auscultatory gap = silent gap:

- **Definition :** period during recording blood pressure by auscultation in which no sounds can be heard but blood passes & can be felt
- Present in some hypertensive patients & mechanism is unknown
- Importance of it : as it may leading to false measurement of systole & diastole
- **How to avoid :**
 - Do Palpatory method at first to know roughly systolic BP
 - Rise pressure in the bag 20 – 30 mmHg above systolic blood pressure known by palpation
 - Do Auscultatory method

❖ Precaution before BP measurement:

1. Patient should rest for 5 -10 min before measurement
2. Arm should be at level of heart e.g. resting on pillow
3. Arm should be supported to avoid isometric exercise which ↑ BP
4. Avoid venous congestion from tight clothing or incomplete deflation from previous measurement
5. Proper size of bag:
 - Width of bag should be 40 % of limb circumference
 - Length of bag should be 80 % of limb circumference
 - Using normal cuff in obese gives false diagnosis of hypertension



6. Be aware of the auscultatory gap
7. Wrap the cuff with the inflatable inner bladder (rubber bag) centered over the area of the brachial artery

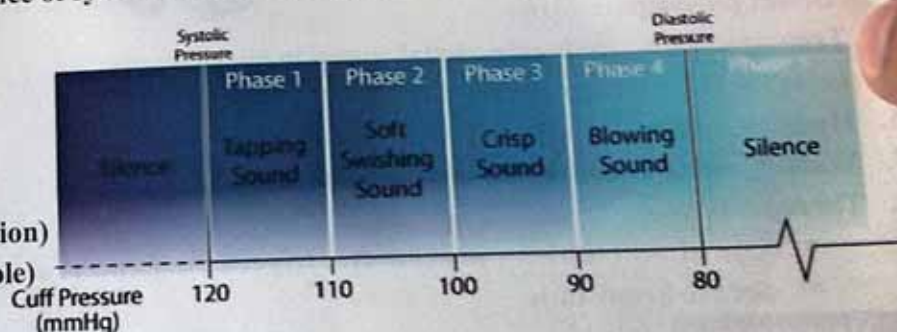
❖ Technique of blood pressure measurement :

1. Precautions must be done
2. Palpate the brachial artery and then wrap the cuff snugly around the patient's arm with the middle of bladder over the brachial artery pulsation (3-5 cm above cubital fossa, medial to the biceps tendon)
3. Inflate the cuff while palpating the radial pulse (to avoid the auscultatory gap) until it's no longer palpable, note the reading (Palpatory method)
4. Deflate the cuff and wait 15-30 seconds before re-inflating
5. Re-inflate steadily with the diaphragm of stethoscope over the brachial artery to a pressure 20-30 mmHg above the level previously determined by palpation
6. Deflate the cuff slowly (2 mm / sec. to avoid error), listening for Korotkoff sounds as they appear (systolic) and then disappear (diastolic) (Auscultatory method)
7. BP is measured in supine position and then ask the patient to stand and re-measure blood pressure after 1 - minutes for postural hypotension (difference of systolic BP between both ≥ 20 mmHg)



❖ Korotkoff sounds (phases) :

1. Phase I: sounds start to appear
2. Phase II : Sounds muffle
3. Phase III : Sounds sharp
4. Phase IV: Sounds muffle (record diastole in hyperdynamic circulation)
5. Phase V: Sounds disappear (record diastole)



❖ Special notes:

1. BP should be taken in Right & Left arms:
 - Normally may be a difference in pressure of 5 -10 mm Hg, subsequent reading should be made on arm with higher pressure.
 - If difference between both arms > 15 mmHg → same causes of unequal pulse.
2. BP in LL is measured by:
 - Application of special large cuff around lower 1/3 of thigh & auscultation of popliteal artery (in popliteal fossa) while patient lies in prone position.
3. Normally systolic BP of LL is more than UL by 10-20 mmHg due to :

• Arteries of LL "femoral" in direct continuity with aorta	• LL muscles are more bulky	• Effect of gravity
--	-----------------------------	---------------------

4. Indication to measure the BP from LL :

A. To diagnose LL ischemia

B. In aortic regurge:

- Systolic BP in LL is $>$ than UL by 60 mmHg (exaggerated normal criteria - Hill's sign)

C. In coarctation of aorta:

- Systolic LL BP is $<$ than UL (opposite normal criteria - reversed Hill's sign)

5. If Volume "Pulse Pressure" is > 60 mmHg, diastolic BP < 60 mmHg then :
 - Diagnose AR & search for its peripheral sign (e.g. Pistol shot)
 - Large pulse volume & water hammer pulse
 - Epigastric pulsation coming from aorta
6. Blood pressure measurement in obese patient
 - by using large sized cuff but if not available we can measure BP by inflating the cuff around the forearm and auscultate radial artery
7. Hypotension :
 - It's decline of systolic blood pressure $< 90-100$ mmHg (supine hypotension)
 - ❖ Causes :
 - Heart failure, Hypovolemia
 - Stenotic lesions, Drugs : diuretics, nitrates, B blockers
 - Addison's disease
 - Orthostatic hypotension
8. Orthostatic hypotension "postural hypotension" & it's measurement.
- ❖ Orthostatic challenge or the tilt test :



❖ قياس الضغط في نفس الوقت وبنفس الجهاز وبنفس لفة الـ cuff مرة والعيان نايم ومرة وهو واقف

- A. Measure the blood pressure in supine position and then ask the patient to stand and re-measure blood pressure after 1-2 minutes
- B. 1-2 minute after standing, about 7-8 ml/kg of blood shift to the lower body $\rightarrow$ $\downarrow$ COP, also increase of circulating catecholamines and systemic vascular resistance occur

C. Normally :	D. Orthostatic hypotension means:
• Pulse rate is increased by about 10 /m & stabilizes after 45 seconds • Systolic blood pressure increase by 3-5 mmHg & stabilizes within 1-2 minutes • Diastolic blood pressure increase by 3-8 mmHg & stabilizes within 1-2 minutes	• Pulse rate is increased at least 30/minute • Systolic blood pressure decreased by ≥ 20 mmHg or • Diastolic blood pressure decreased by ≥ 10 mmHg

E. Causes of Orthostatic hypotension:

- Antihypertensive drugs e.g. ganglion blocker,
- Autonomic neuropathy e.g. DM
- Addison's disease
- Lumbar sympathectomy
- Prolonged recumbency, Elderly patient

4. Respiratory movements

	1. Rate	2. Rhythm	3. Depth	4. Type of breathing
▪ Normal  → Count respiratory rate while you are simulating counting the pulse rate to distract the attention of the patient.	• 12 – 20 /min (pulse : respiration ratio = 4 : 1)	• Regular	• Full expansion of the chest wall with full relaxation i.e. average	• Females : thoraco-abdominal • Males : abdomino-thoracic
▪ Abnormally	▪ For more details : see chest chapter			

II. General overview (A, B, C, D, E)

❖ Appearance, Built & height, Consciousness, Colours, Decubitus & facial Expression.

1. Appearance

- Healthy, ill, toxic, depressed, elation, anxious, irritable ... etc.

2. Built & height

1. Built by inspection, (weight by weighing scale)

- Average built or over built or Under built

- Body mass index : $\frac{\text{weight in Kg}}{(\text{height in m})^2}$

❖ Causes of obesity & underweight: see later in endocrinology chapter.

Category	Body mass index = BMI
▪ Underweight; malnutrition	Below 18.5
▪ Average normal weight	18.5 – 25
▪ Overweight	25 – 30
❖ Classification of obesity :	
▪ Class I : Mild obesity	30 – 35
▪ Class II: Moderate obesity	35 – 40
▪ Class III : Marked (Morbid) obesity	> 40

2. Height :

- Average or giant or dwarf

A. The height and span are equal (proportionate) :

- The height : distance from occiput to heel in upright position
- Span : the distance between the tip of middle fingers with outstretched hands

B. Lower and upper segment are usually equal :

- Lower segment : distance from symphysis pubis to the floor
- Upper segment : distance from the occiput to the symphysis pubis

❖ Causes of gigantism "tall stature" & dwarfism "stunted growth = short stature": see later in endocrinology chapter.



3. Consciousness

❖ For more details : see neurology chapter

- Patient is conscious cooperative, well oriented to time, place & person, average memory, mentality (intelligence) mood (affect)

• Conscious (mentality) is affected in :

- Neurological & psychiatric cases
- Major organ failure :

▪ Respiratory failure (CO₂ narcosis) ▪ Hepatic encephalopathy ▪ Uremic encephalopathy

4. Colours = Complexion

A. Pigmentation	B. Pallor	C. Sclera "yellow & blue"
D. Cyanosis	E. Rash & vascular lesions	

A. Pigmentation:

❖ Causes of generalized pigmentation	❖ Causes of localized pigmentation
 1. Familial & racial 2. Primary Addison's diseases 3. Primary biliary cirrhosis 4. Pituitary Cushing disease 5. Pellagra 6. Neurofibromatosis 7. Hemochromatosis 8. DM 9. Skin diseases 10. Leprosy	  • As butterfly area of the face : butterfly rash: A. <u>Red</u> : • Malar rash : MS with PH (reflex VD or compression of sympathetic chain) • Cushing syndrome , Myxedema , alcoholics normal variant • Discoid rash : SLE B. <u>Brown</u> : • Pellagra • Chloasma gravidarum : in pregnancy

B. Pallor :

1. **Definition** : decrease visibility of oxyhemoglobin

2. **Detected in**:

A. Mucous membrane:

- Inner side of lips (slightly pull the lower lip outward & look for the colour inside)
- Conjunctiva : but not in Egypt because of endemic trachoma which produces whitish membrane in eyelid.

B. Tongue (more accurate)

C. Palmer crease of hand, nail



3. **Causes**:

1. Decrease blood flow to superficial vessels : low CO, shock, syncope, HF, toxemia (SBE)
2. Anemia
3. Edema (mask color of melanin & HB) : nephrotic syndrome
4. Others: myxedema, lymphedema, albinism

C. Sclera "Yellow & Blue"

Yellow: jaundice	Blue :
 ▪ Yellowish discoloration of skin & mm due to total bilirubin more than 3 mg % i.e. manifest jaundice ▪ Subclinical jaundice : 1-3 mg% ▪ Normally total bilirubin : 0.2 - 1 mg %	 ▪ Thin sclera showing choroidal pigment beneath
❖ Deposited in: ○ Sclera: because sclera is rich in <u>elastin</u> & elastin has <u>high affinity</u> to bilirubin & sclera is <u>white</u>. → Examined in lower fornix of the eye; asking the patient to look upwards and facing the day light (as yellow light lamps may produce artificial jaundice) ○ Soft palate	❖ Causes : ○ Normal variant ○ Congenital glaucoma "Buphthalmos" ○ Osteogenesis imperfecta ○ TB

❖ What are the types of Jaundice?

	Hemolytic Jaundice	Obstructive jaundice	Hepatocellular
Cause	Excess hemolysis of RBCs → ↑ hemobilirubin	Obstruction of bile ducts → ↑ cholebilirubin	Hepatitis, cirrhosis → ↑ both hemobilirubin & cholebilirubin
Sclera	Lemon yellow jaundice	Olive green jaundice	Orange yellow jaundice
Stool	Dark stool	Pale stool	Pale stool
Urine	Normal urine colour which darkens in standing (acholuric jaundice)	Dark urine	Dark urine
Others	Pallor, leg ulcers, ↑ liver & spleen	Biliary pain, pruritus & bradycardia	Clinical picture of liver cell failure

❖ What are the differential diagnoses of jaundice?

- Normal yellow sclera : in Negroes (Negros normally cyano-icterus)
 - Hypercarotenemia
- Causes :
 - Increase intake of carotene in vegetables
 - DM
 - Vitamin A deficiency
 - Myxedema, liver cell failure
 - Sites : It appears in palm, soles & face, but not in the sclera and mucous membranes
 - Xanthomatosis: see later.
 - Chronic Renal Failure : retention of yellow pigments (urochromogen) superimposed on pallor of anemia (dirty yellow)
 - Drugs : atebrine, miracil-D, picric acid toxicity



D. Cyanosis :

Normal reduced HB:
2.1-2.4 gm /dl

- ❖ **Definition:** bluish discoloration of skin & mucous membrane (mm)
- ❖ **Etiology :**
 - ↑ level of reduced HB in capillaries > 5 gm /dl or abnormal HB in capillaries
- ❖ **N.B. :**
 - Cyanosis never occurs in severe anemia : incompatible with life
 - Cyanosis appears easily in: polycythemia

❖ Types of cyanosis:

- Chemical cyanosis: "false cyanosis"
 - Definition : Abnormal HB simulates reduced HB
 - Clinically: simulate central cyanosis
 - Causes:
 - Met-hemoglobinemia (nitrite treatment)
 - Sulf-hemoglobinemia (sulfonamide treatment)
 - Diagnosed by: spectroscopy
- Differential cyanosis
 - Definition : Cyanosis in LL only (absent in UL)
 - Causes :
 - PDA with pulmonary hypertension (Eisenmenger PDA)
 - PDA with preductal (infantile) aortic cortication



	3. Central Cyanosis = Hypoxic Hypoxia	4. Peripheral Cyanosis = Stagnant hypoxia
		
1. Definition	• Blood pumped in aorta contains more than 5 gm % reduced HB	• Stagnation of blood in peripheral circulation leads to extraction of more O ₂
2. Causes	• Cardiogenic acute pulmonary edema & Polycythemia • Congenital Heart disease with right to left shunts: Eisenmenger's, F3, F4 • Chest : COPD, IPF, pulmonary embolism • Asphyxia, high Altitude • Opening of Pulmonary A-V shunts : liver cirrhosis & failure	• Low Cardiac output : shock • Congestive heart failure • Cold exposure • Arterial & venous diseases : Raynaud's & thrombosis
3. Site	• Tongue (under surface ± upper S.) • Generalized : inner aspect of lips, tips of fingers, toes, nose & lobule of ear	• Generalized : inner aspect of lips, tips of fingers, toes, nose & lobule of ear <i>NOT in the tongue</i>
4. Hand	• Warm due to hypoxia (VD)	• Cold due to peripheral VC
5. Warming	• No effect	• Cyanosis improves
6. Clubbing	• Present (blue clubbing)	• absent
7. O ₂ Saturation	• Low < 80%	• Normal (95 - 98%)
8. O ₂ TTT	• If lung disease : improves cyanosis • If congenital HD : no effect	• No effect

❖ Why are lips not preferred as a site for cyanosis?

- Smokers : blue lips
- Negros normally cyano-icteric tinge

❖ Which part of the tongue do you look at for cyanosis ?

- You look at undersurface from forwards

? Why?

- Undersurface : as papillae coating the upper surface
- Forwards : as the veins posteriorly

❖ What are the causes of combined central & peripheral cyanosis ?

☞ Cardiogenic acute pulmonary edema & Polycythemia

❖ How to unmask peripheral cyanosis in a patient with combined central & peripheral cyanosis ?

☞ The patient with peripheral cyanosis has cold hands so warming hands unmasks it.

❖ Why doesn't peripheral cyanosis affect the tongue?

• Because peripheral cyanosis is caused by blood stagnation which can't occur in the tongue being:

- Active mobile organ → VD
- Warm oral cavity → VD
- Devoid of vasoconstrictor sympathetic supply (No autonomic fibers)
- Well perfused as double blood supply of the tongue

❖ When does not central cyanosis appear in tongue?

• In cases of differential cyanosis

❖ When does peripheral cyanosis appear in tongue?

• SVC obstruction

• Lingual vein thrombosis : localized cyanosis in the tongue

E. Vascular lesions & rashes :

1. Spider naevi:

- They consist of central dilated arteriole with radiating capillaries resembling spider's legs.
- Mechanism : unknown but 2 theories : excess estrogen theory & vasodilator materials theory
- Compression of the central arteriole, causes blanching of the whole lesion,
- Releasing of the pressure , the blood refill the leg's of spider
- They are found in the distribution of the SVC (face, neck, UL & upper chest above the nipple line)
- Size : variable (from pinhead to about 0.5 cm)
- Non-specific ; Causes : may be found in normal people, pregnancy ,thyrotoxicosis & LCF, vitamin B12 deficiency
- Confirmed by :
 - Pin – head test: Central pressure = blanching
 - Glass Slide test = pulsations



2. Erythema:

A. Erythema marginatum	B. Erythema nodosum	C. Erythema multiforme
• Painless non tender red spots over the trunk & proximal extremities with serpiginous margin • In Rheumatic fever (major criteria)	• Painful red indurated nodules on the shin of tibia, thighs, knees, extensor surface of arms & forearms • Caused by: ◦ Primary TB ◦ Sulfonamides, Streptococcal infection, Sarcoidosis	• Red ring with central vesicle or hyperpigmented known as Target iris lesion • Painful if there is mucous membrane involvement • Caused by : ◦ Sulfonamides, HSV ◦ Mycoplasma pneumonia

Others : = Differential diagnosis of spider nevi :

	Level	Pruritus	Effect of Pressure	Pulsating	Site
• Spider naevi	Raised	Not	Fading	Pulsate	SVC distribution
3. Insect bite	Raised	Itchy	Fading	Not	Exposed areas
4. Purpura	Not	Not	Not fade	Not	Common in LL & lower abdomen
5. Campbell de Morgan spots = Cherry (senile) angioma	Raised	Not	Not fade	Not	Trunk & extremities
6. Venous star	Variable	±	Increased وضوحاً	Not	LL
7. Hereditary hemorrhagic telangiectasia (HHT) = (Osler-Weber-Rendu syndrome)	Raised	Not	Fading	±	• Skin • On mucous membrane e.g. lips & tongue • Organs

• Insect bite	• Campbell de Morgan spots	• Venous star	• HHT

5. Decubitus

❖ Normal : no special decubiti; the patient is lying comfortable in bed

❖ Abnormally :

1. Sitting up : Bronchiectasis, massive ascites
2. Prayer position: the patient prefers to lean forward :



○ Posture of drainage in bronchiectasis, pericardial effusion, aortic aneurysm & acute pancreatitis

3. Orthopnea: dyspnea on lying flat improves on semisitting, using high pillows



○ Causes: Left side heart failure, MS, pregnancy, tense ascites & status asthmatics, severe emphysema.

○ N.B. : orthopnea is not pathognomonic to left side heart failure.

4. Platypnea : dyspnea in the erect position relieved by lying flat e.g. in hepato-renal syndrome

5. Trepopnea (Lateral decubitus) : patient prefers to lie on one side



○ On the affected side : pleurisy , lung abscess (postural drainage)

○ On healthy side in lung collapse

6. Squatting position : in Fallot's tetralogy



6. Facial Expression

❖ Facial expression of patient : normal (don't mention it) or

❖ Abnormally :



- Acromegaly :
Ape face : coarse features, enlarged ears, nose, lips, tongue



- Cushing syndrome :
Moon face, erythema (plethoric cheeks) with acne, hirsutism (moustache)

○ Chronic illness: Emaciated; wasted temporalis.

☞ Other facial expression, in each chapter with the related cases.

III. Systemic overview

1. Head:

A. Head

1. Skull e.g. shape e.g. acromegaly : square shape
2. Hair e.g. silky hair in LCF or sparse hair in myxedema

B. Eye :

1. Hair loss in outer 1/3 in myxedema & leprosy
2. Xanthelasma is a yellowish eruption at the inner side of the eyelids & periorbital skin associated with hypercholesterolemia.
3. Exophthalmos in thyrotoxicosis, cavernous sinus thrombosis, orbital tumor.
4. Eye lid :
 - Retraction : thyrotoxicosis
 - Ptosis (unilateral or bilateral) : see neurology.
 - Pupil: see neurology
 - Puffiness of eye lid: see discussion
 - Eye movement : see neurology
5. Sclera : jaundice
6. Lens : subluxation in Marfan's syndrome

C. Nose :

1. Size : enlarged in acromegaly, myxedema
2. Redness of the in SLE
3. Sulphur granules in riboflavin deficiency
4. Working ala nasi in respiratory failure
5. Nasolabial fold obliterated in facial palsy

D. Butterfly area = local pigmentation :

- Red or Brown : causes : see before .
- Petichae : in SBE , differentiated from :
 - Spider naevi in liver cell Failure : fade on pressure
 - Insect bite "flea" : itchy, generalized

E. The mouth :

1. Angular stomatitis in iron deficiency anemia & ariboflavinosis (↓Vitamin B2)
2. Lips : pallor, cyanosis (Negros normally cyano-icterus), chielosis (dry cracked in Ariboflavinosis)
3. Teeth : separation of the lower teeth may occur in acromegaly, loosening of teeth in DM
4. Tongue :

a) Size	• Large: acromegaly, myxedema, cretinism	• Small (LMNL)
b) Color	• Pale in anemia, blue in central cyanosis, slate colored in Addison disease, brown in smoking, black with iron therapy	
c) Papillae	• Atrophy (red glazed tongue): in iron deficiency anemia, B12 deficiency, pellagra, liver cirrhosis, DM & anticancerous drugs.	• Enlargement (strawberry tongue): in scarlet fever
d) Surface	• Dry tongue : in mouth breathers, dehydration • Coated tongue as in typhoid fever	
e) Movement:	Fasciculations, tremors, power, deviation : see neurology.	

5. Buccal mucosa : pigmentation in Addison's disease

6. Palate : high arched palate in Marfan Syndrome

7. Uvula : see neurology

8. Odour of the mouth = causes of bad odour :

Acetone	DKA
Ammoniacal	Renal failure
Alcohol	Alcoholol abuser
Halitosis	Suppurative lung syndrome (SLS), cancer esophagus, bad oral hygiene
Fetor hepaticus	LCF

F. Parotid gland : parotitis enlargement

- Epidemic → painful tender unilateral as in mumps
- Others: TB, Leukemia, Sjogren's syndrome
- Endemic → painless bilateral as in hypoalbuminemia as in cirrhosis & nephrotic syndrome

- Acromegaly



- Thyrotoxicosis



- Myxedema



- Silky Hair



- Xanthelasma



- Angular Stomatitis



- Red Glazed Tongue



- Strawberry Tongue



- Buccal Mucosa : Pigmentation



- Dry Tongue



- Coated Tongue



- High Arched Palate



- Parotid Enlargement



- Ptosis



- Puffy Eyelids



2. Neck :

1. **Skin:** for carbuncle on the back in DM →



2. **Lymph Node:** if enlarged continue lymphadenopathy sheet, see LN chapter.
3. **Pulsation:** arterial & venous

A. Carotid Pulsation :

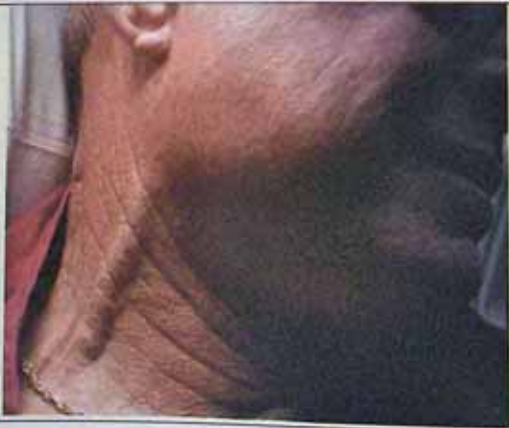


- Inspection : visible arterial pulsation:
 - May be seen in thin people
 - Same causes of big pulse pressure especially AR: " Corrigan's signs"
- Palpation :
 - 1) Palpable carotid pulsation or absent carotid pulsation (i.e. Berry's sign) as in thyroid malignancy.
 - 2) Volume
 - 3) In place (felt against carotid tubercle of C6) or displaced
 - 4) Special Character (pulsus bisferiens in Double Aortic lesion & IHSS; idiopathic hypertrophic subaortic stenosis)
- 5) Carotid Thrill, Timing : Systolic
 - ⚡ Accompanied by thrill over the base of the heart :
 - AS, PS, PDA
 - ⚡ No thrill over the base of the heart " initiated from carotids" "carotid shudder" :
 - Carotid aneurysm, Peripheral signs of AR
 - Other causes of big pulse pressure
- 6) Equality on both sides in pulse volume. (DO NOT examine both carotid in the same time)
 - Auscultation: carotid bruit in carotid stenosis

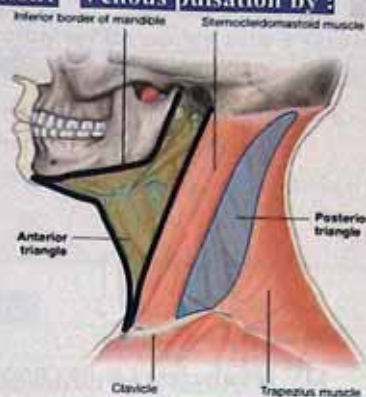
B. Neck Veins : see later

4. **Trachea:** central or shifted. See chest
5. **Thyroid:** see endocrinology.
 - Inspection and describe
 - Palpation : for systolic thrill in thyrotoxicosis
 - Percussion : upper border of sternum (for retrosternal extension)
 - Auscultate : for systolic bruit (increased vascularity in thyrotoxicosis)

Neck veins

External jugular vein	Internal jugular vein
❖ Surface anatomy:	
▪ Line drawn from angle of mandible to mid-clavicular point (junction of medial 1/3 & lateral 2/3)	▪ Line drawn from lobule of the ear (midway between angle of mandible & mastoid process) to medial end of the clavicle sternoclavicular joint
	

1) Is it arterial pulsation or venous pulsation? Venous pulsation by:



A. Arterial pulsation = carotid arteries

B. Venous pulsation = neck veins

- Medial to sternomastoid, in Anterior triangle of neck
- More apparent in upper part of neck
- NO waves (one jerk)
- Synchronous with heart beat
- Better felt than seen
- NOT obliterated by Pressure
- NOT affected by Posture
- NOT affected by REspiration
- NO hepato-jugular REflux

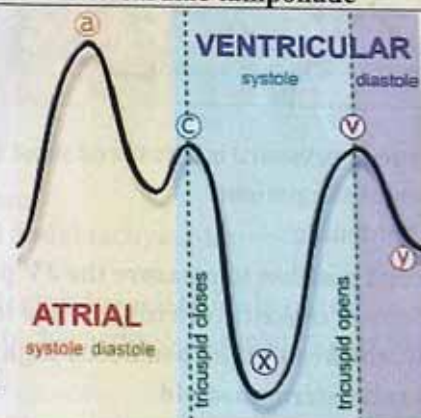
- Lateral to sternomastoid, in posterior triangle of neck
- More apparent in Lower part of neck
- Wavy (a & v waves)
- Asynchronous with heart beat (a wave)
- Better seen than felt
- Obliterated by pressure
- Change with posture
- Change with inspiration (↓)
- Hepato-jugular reflux : (↑)



- ❖ Hepato-jugular reflux; one minute abdominal compression test:
- Mild pressure on liver or abdomen leads to elevation of upper level of vein with no affection of artery
- It is positive in Right ventricular failure, TR, TS, Constrictive pericarditis & cardiac tamponade

2) What are the waves of neck veins?

= jugular venous pulse, physiology of the waves:



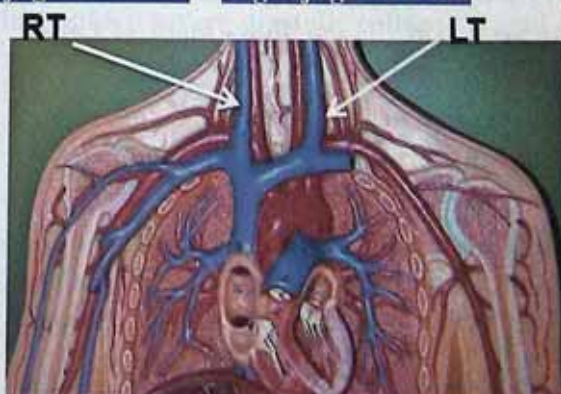
- ⇒ Normal JVP = 0-4 cm H₂O
- ⇒ The waves of JVP act as manometer which reflects pressure changes in Right atrium
- ⇒ Consists of 3 positive (A, C & V) & 2 negatives (X & Y)
- ⇒ The A wave is normally the highest wave
- ⇒ The X wave is usually deeper than the Y wave
- ⇒ Radial pulsation is preferred for timing of the venous pulsation

❖ Wave	❖ Represents	❖ Timing
▪ a wave	○ Right atrial contraction	• Presystolic wave
▪ c wave	○ Upward bulge of tricuspid valve into right atrium at start of ventricular contraction ○ Or Transmitted from adjacent carotids at the onset of ventricular systole	
▪ x descent	○ Right atrial relaxation (normal systolic collapse)	• Systolic wave
▪ v wave	○ Right atrial filling from the venous blood	• Systolic
▪ y descent	○ Right atrial emptying to right ventricle (diastolic collapse)	• Diastolic

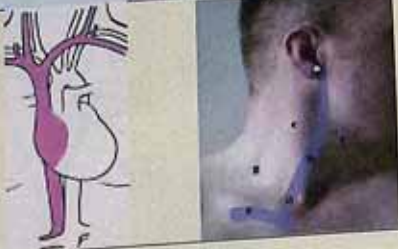
3) Which side do you will examine, right or left jugular veins?

Right jugular veins

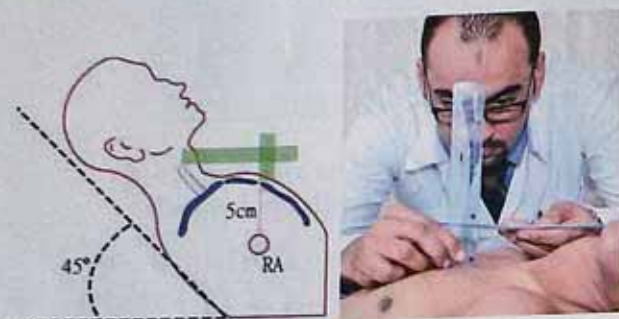
- Right is preferred as Left sided ones may be present falsely prominent (high) as the left innominate vein (Brachio-cephalic) may be compressed by the arch of aorta.



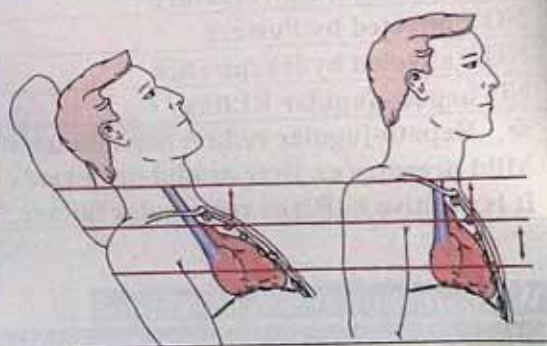
4) Is it external or internal jugular veins? Pulsation of IJV

❖ External jugular vein	❖ Internal jugular veins
 External jugular SCM muscle Clavicle Internal jugular	
- The EJV is more superficial and prominent but we don't examine it because - It's prone to kinking and partial obstruction as it traverses the deep fascia of the neck. - It has a tortuous course	■ Examination: - IJV is valveless i.e. has no venous valves, so it reflects exactly the changes in right atrium - It has the most direct channel (direct continuity) to the right atrium - When the pressure in the IJV is elevated you can see a diffuse pulsation not the vein itself. (SO WE ARE EXAMINING THE PULSATION NOT THE VEIN ITSELF)

5) Why & How to measure the jugular venous pressure? To know either Congested or not congested



If the pulsation not visible



More elevation up to 60° or 90°

A. The venous pressure is measured most accurately by manometry

B. Position of the patient:

1. Good lightening
2. No special position to measure the JV pressure, the aim to adjust the head of the bed so as to maximize the JV pulsations & make them visible in the lower half of the neck (above the clavicle, but below the jaw).
3. Usually, the head of the bed needs slight elevation; 45° from the horizontal, and the face is directed to the left side to relax sternomastoid
4. When venous pressure is severely increased (severely congested neck vein), more elevation up to 60° or even 90° may be required to make waveform (pulsations) is clearly visible.

C. Measure the vertical distance between the two points :

- a) Put ruler vertically on the manubrio-sternal angle of Louis
- b) Put ruler rectangular on highest point of internal jugular pulsation

D. Possibilities after measurement :

- a) Normally, the JVP is visible 2-4 cm above the sternal angle i.e. not congested
- b) IF abnormally above 4 cm i.e. congested; comment on the following :

1. **Congested Pulsating neck vein** cm above angle of Lewis

2. **Relation to respiration:**

- Either empty with inspiration or
- Expiratory filling & inspiratory collapse in emphysema.

3. **Hepato-jugular reflux** : +ve or -ve

4. **Timing** : (Radial pulsation is preferred for timing of the venous pulsation)

- Either systolic collapse or systolic expansion
- All causes are systolic collapse except { AF, TR & cannon a waves → Systolic expansion }

Normal neck veins:
pulsating not congested,
empty with inspiration
with systolic collapse

- ❖ Example for a comment on neck (with a case of AF):
- ♥ Congested pulsating neck veins Above angle of Lewis.
 - ♥ Congestion decreases with inspiration
 - ♥ Positive hepato-jugular reflux
 - ♥ There is systolic expansion with absent a wave
 - ♥ CVP = ... cm

6) How to measure central venous pressure?

- Pressure in center of Right atrium as measured by a catheter
- $CVP = JVP + 5 \text{ cm} = \dots \text{ cm H}_2\text{O}$ {Normal CVP = 7 – 10 cm}

7) What are the causes of congested neck veins?

A. Pulsating	B. Non pulsating
• ↑ Right atrial Pressure : right sided HF, TR • ↑ Intrathoracic Pressure: physiological (cough, straining), pneumothorax, emphysema • ↑ Intraabdominal Pressure : tense ascites • ↑ Blood volume: anemia, pregnancy, fluid overload	• Severe Right sided failure : severe congested neck veins "can't see upper border" • Complete SVC obstruction : thrombosis & mediastinal syndrome • Pericardial diseases : Pericardial effusion & constrictive pericarditis

8) What are the common abnormalities in jugular venous pulse? = abnormal waves

1. Absent a wave in:	2. Giant (prominent) a wave (forceful atrial contraction) in :	3. Cannon a waves (synchronous atrial & ventricular contraction) in:
• AF (no atrial contraction)	• TS • PS • Pulmonary hypertension	⚡ Regular: ○ Nodal rhythm ○ Paroxysmal nodal tachycardia ⚡ Occasionally : ○ Atrio-ventricular dissociation e.g. paroxysmal ventricular tachycardia & complete heart block
4. Causes of giant v wave or causes of obliteration of x descent? • Systolic expansion; AF, TR		
5. Prominent x descent in:	6. Prominent y descent in :	7. Attenuated or absent y descent in :
Constrictive pericarditis Pericardial effusion	Constrictive pericarditis	Pericardial effusion

8. Neck veins in constrictive pericarditis show 2 signs:

1. Kussmaul sign: congested neck veins showing inspiratory filling (Reverse of the normal)
2. Friedrich 's sign: congested Neck veins showing sudden severe diastolic collapse (rapid & deep Y decent)

3. Upper limb, examine BOTH sides

1. Skin of the hand:

- A. Elasticity : *tense in scleroderma & edema, loose in loss of weight*
- C. Dry skin : myxedema

- B. Thickness : *thick in acromegaly, myxedema, atrophy in peripheral neuritis*
- D. Excessive sweating in thyrotoxicosis & neurosis

2. Shape of the hand :

- A. Square in cretinism
- B. Spade like hand in acromegaly (large & broad)
- C. Arachnodactyly (spider finger) long tapering fingers as in Marfan syndrome
- D. Dupuytren's contracture: "palmar fibromatosis" : fibrosis of palmar aponeurosis with flexed inner 2 or 3 fingers; Causes :

- Idiopathic : familial
- Secondary : DM, alcoholism & alcoholic liver cirrhosis

3. LN of the hand : see later

4. Pulse & blood Pressure (see before)

5. Palm:

- A. Palmar erythema: definition :

- Erythema in thenar & hypothena eminences and over the pulps of fingers **with central pallor**
- B. Pallor in palmar creases in anemia

- C. Sweating in palm & dorsum & warm hand in: Thyrotoxicosis

- D. Sweating in palm only & cold hand in : neurosis

- 6. Nail : normally : shiny, convex in lateral view and obtuse nail bed angle; 160 degrees

❖ TCKSLY : تكسلي

- A. Trophic changes: in the form of loss of luster, striated, brittle nails as in polyneuropathy, iron deficiency & chronic ischemia.
- B. Terry's nail : distal half is brown while proximal half is white-pink in LCF & CRF
- C. Cyanosis (central or peripheral) , Clubbing, Capillary pulsation in AI
- D. Koilonychia (nail spooning): flat then concave in iron deficiency anemia
- E. Splinter Hemorrhage in SBE
- F. Leukonychia: white nail in hypoalbuminemia as in LCF, Nephrotic syndrome & normal
- G. Yellow nail syndrome : yellow in colour + peripheral oedema + bronchiectasis + pleural effusion)

7. Nodules :

- A. Osler's nodes in pulps of finger in SBE
- B. In osteoarthritis : Herberden's nodules: in DIP , Bouchard nodules : in PIP.
- C. Subcutaneous nodules in rheumatic fever & Rheumatoid arthritis
- D. Gouty tophi

8. Clubbing : see later.

9. Colors :

- Yellowish brown in chronic renal failure.
- Reddish blue or purple discoloration of the skin that doesn't blanch on pressure :

- Petechiae : less than 2 mm
- Purpura: 2 mm to 1 cm
- Ecchymosis : more than 1 cm

10. Trophic changes:

- Ulcers, Loss of Hair & Brittle nail

11. Tremors: اطلب من العيان بغض عينه

- ✂ Fine tremors : use a paper sheet

- ✂ Flapping tremors = coarse tremors = asterixis : rapid flexion & extension at the wrist & MCP joints
- How to examine : outstretched arms, supported & fanning of fingers
- Induced by sudden dorsiflexion of the pronated wrist.

12. Joints & Deformity: See later in each system (neurology, rheumatology , ...etc.)

13. Muscles & nerves : Sensation, Muscle wasting , Movement, power & Reflexes; see neurology.

❖ Causes of palmer erythema :

- P : Polycythemia, Pregnancy
- A : Alcohol intake, Autoimmune (RA,SLE)
- L : Liver cell failure , Leukemia
- M : Medications (CCPs, Corticosteroids), Miscellaneous (Thyrotoxicosis)



Flapping tremors



Fine tremors

4. Lower limb, examine BOTH sides

A. Odema:

- Technique : by 1 minute pressure test
- Sites : behind medial malleolus , chin of tibia & dorsum of foot
- Other sites : sacrum over the back in bedridden patients

❖ After pressure look for:

- 1) Tenderness : Tender or not
- 2) Type : pitting or non-pitting (non-pitting in myxedema)
- 3) Laterality : bilateral or unilateral
- 4) Symmetrical or not by tape.
- 5) Extent :



Peau d'orange

❖ If below knee :	❖ If above knee:
○ Above ankle ○ Mid leg (medial side of leg) ○ Below knee (tibial tuberosity)	○ Pinching test over the thigh ○ Peau d'orange over the anterior abdominal wall

6) General assessment of the patient with lower limb edema :

- Examine the liver & heart
- Examine back for sacral oedema
- Examine serous sacs for ascites, pericardial or pleural effusion

B. Rest of examination as upper limb but:

- In clubbing we detect it by inspection only
- No tremors

Arachnodactyly



Dupuytren's contracture



Palmar erythema



Trophic changes in the nail



Terry's nail



Koilonychia (nail spooning)



Splinter Hemorrhage



Yellow nail syndrome



Leukonychia



Osler's nodes



Herberden's nodules



Bouchard nodules



Gouty tophi



Petechiae & purpura



Ecchymosis



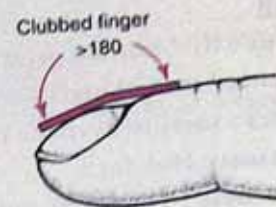
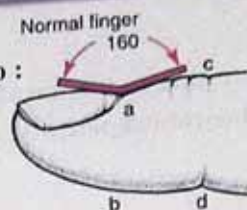
Clubbing

❖ Definition & mechanism:

❖ Proliferation of CT under nail bed in response to :

1. Chronic hypoxia : blue clubbing = cyanotic
2. Chronic toxemia : pale clubbing = acyanotic
3. Other mechanisms: irritation, immune.

❖ Causes of clubbing :



Other mechanisms: irritation, immune.			
Causes of clubbing :			
A. Chronic hypoxia : blue clubbing = cyanotic		B. Chronic toxemia : pale clubbing = acyanotic	
			
❖ Hypoxia leads to clubbing within several months and even years		❖ Toxemia leads to clubbing within few weeks	
Cardiac			
• Congenital cyanotic Heart disease with Right to Left shunts: Eisenmenger's, F3 & F4		• Subacute bacterial endocarditis	
Chest			
• COPD; clubbing usually mild – marked in associated bronchiectasis • Interstitial pulmonary fibrosis		• Suppurative lung syndromes : lung abscess , bronchiectasis, empyema & cystic fibrosis • Fibroid type of pulmonary TB • Bronchogenic carcinoma • Pleural methothelioma	
Abdominal			
• Liver cirrhosis especially primary biliary cirrhosis		• Ulcerative colitis & chrohn's disease • Familial polyposis & Bilharzial polyposis • Steatorrhea	

C. Occupational : shoe makers , carpenters (usually in thumb & index)

D. Miscellaneous :

- Familial : 5 % of cases
- Thyroid acropachy: in 1 % of patient with Graves : clubbing + exophthalmos + pretibial myxedema
- Primary hypertrophic osteoarthropathy; Pachydermoperiostosis : Characterized by pachydermia(thickening of the skin), periostosis (excessive bone formation) and finger clubbing.

❖ What are the causes of reversible clubbing?

- Pleural methothelioma, endocarditis, empyema

❖ What are the causes of unilateral clubbing?

- As same causes of unequal pulse

❖ What are the causes of clubbing in LL only (differential clubbing) ?

- Same causes of differential cyanosis

❖ Degrees "grades" :



Parrot peak

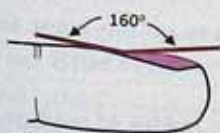


Drum stick

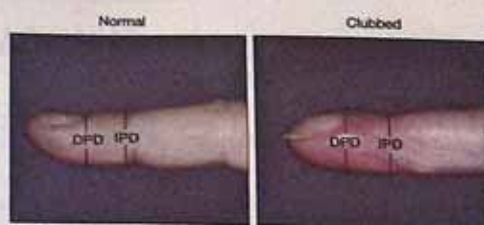
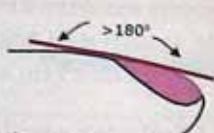
1. 1st degree: obliterated nail bed (window or Schamroth's sign) & fluctuation is present.
 2. 2nd degree: parrot peak i.e. ↑ of curvature of the nail in its longitudinal axis.
 3. 3rd degree: drum stick i.e. swelling of the pulp of the finger in all its dimension.
 4. 4th degree: drum stick + pulmonary hypertrophic oesthoarthropathy i.e. tender thickening of ends of long bones " radius & ulna", it is due to subperiosteal new bone formation, so it's an X-ray finding.
- ❖ N.B. if intermediate degree say : from ... passing to ... e.g. from parrot peak passing to drum sick.

❖ Diagnosis of clubbing :

Normal



Clubbing



1. Inspection from the side:

A. Lovibond angle : the angle between the nail base and it's adjacent skin

- Normal Lovibond angle: is 160°
- Abnormally : clubbing : obliteration of the Lovibond angle i.e. ≥ 180 degree according to shape , you can determine the degree .

B. Phalangeal depth ratio :

- Normally : interphalangeal depth (IPD) is more than distal phalangeal depth (DPD)
- Abnormally : clubbing : the ratio is altered

Schamroth sign

Normal



Clubbed



Fluctuation test



2. Window sign or Schamroth's sign: ask the patient to approximate the dorsal surface of both finger nail opposite each other, normally a window is seen, absence of this window suggests 1st degree of clubbing

3. Fluctuation test :

♥ تسند أصابع العيان بال 2 thumbs وتجيح بال 2 indices وتدوس على زاويتي اتصال الظفر باللحم في نفس الوقت كذا مرة

- Place the finger on the pulp of the examiner's two thumb and hold in this position by gentle pressure with the tips of the middle fingers over the proximal interphalangeal joint
 - Then palpate the finger over the base of the nail with tips of examiner's two index fingers.
 - When fluctuation is marked, palpation of the nail itself may give the impression that is floating free in its bed.
4. Floating test: press on the tip of the nail → floatation of the part under the skin.

❖ Differential Diagnosis of clubbing = causes of pseudo-clubbing:

☞ Definition:

- Over curvature of the nails in both the longitudinal and transverse axes, with preservation of a normal nail bed angle

☞ Causes of pseudo- clubbing:

▪ Abscesses of the terminal pulp space	▪ HyperParathyroidism
▪ Chronic Paronychia	▪ Chronic renal failure
▪ Subungual hematoma	▪ Sarcoidosis, Scleroderma

❖ Finally : comment on the following in a case of clubbing

- ♥ Present or not
- ♥ Distribution : generalized (UL & LL) or differential (LL only)
- ♥ Degree : قول الوصف متقولش الرقم لان هناك اختلاف في الأرقام
- ♥ Type : cyanotic (blue) or toxemia (pale)
 - Cyanotic clubbing : cyanosis in tongue & hands & warm hands

CLINICAL DISCUSSION

❖ What are the Causes of edema ?

A. Generalized edema (bilateral):	B. Local edema (unilateral) = causes of tender edema = causes of edema in one UL or LL:
1. Cardiac edema : congestive HF, pericardial effusion 2. Renal edema : nephritic or nephrotic syndromes 3. Hepatic edema : liver cell failure 4. Nutritional edema : severe nutritional deficiency or malabsorption syndrome 5. Angioneurotic edema : allergic edema	1. Inflammatory e.g. cellulitis: signs of inflammation as redness, hotness & tenderness 2. Lymphatic obstruction e.g. lymphedema : edema is non pitting 3. Venous obstruction e.g. D.V.T: dilated veins will be present.

❖ What are the Causes of pitting & non pitting oedema ?

▪ Soft pitting (low protein content) : renal, cardiac & hepatic	▪ Hard non pitting (high protein content): Lymphatic obstruction & myxedema
---	--

❖ What are the Causes of tremors ?

A. Fine (↓ amplitude, ↑ frequency):	B. Coarse (↑ amplitude, ↓ frequency):
▪ Anxiety "neurosis", fatigue & excess coffee, tobacco ▪ Thyrotoxicosis "fine rapid tremors " ▪ Toxic: alcohol, mercury, cocaine ▪ β_2 agonist ▪ Familial , Fatigue, Senile	▪ Flapping tremors "Astrexis" : Metabolic encephalopathy especially organ failure as liver, renal, respiratory failure. ▪ Parkinsonism: coarse slow static tremors (at rest & disappear by movement) ▪ Cerebellar ataxia: Intension kinetic tremors; during movement

❖ What are the Causes of puffy eye lid?

❖ Edema : ▪ Generalized : Renal diseases (nephrotic or nephritic), as a part of generalized oedema (rare) ▪ Local : angio-neurotic oedema	❖ Increase : ↑ ▪ ↑ intrathoracic pressure in Chronic cough ▪ ↑ venous pressure : SVC obstruction , congestive HF, pericardial effusion, constrictive pericarditis
❖ Deposition : myxedema	

❖ What are the hazards of smoking ?	❖ What are the hazards of alcohol consumption ?
▪ Chest: Chronic bronchitis, COPD, Bronchogenic carcinoma & Cancer: lip, tongue, pharynx & larynx ▪ CVS: Ischemic heart disease, arrhythmias , stoke , Peripheral vascular disease ▪ GIT: Peptic ulcer & gastritis, Cancer: esophagus, stomach, bladder	▪ CNS: Wernicke-Korsakoff syndrome, Polyneuropathy & Tremors ▪ CVS: Cardiomyopathy ▪ GIT: Alcoholic cirrhosis, Pancreatitis, peptic ulcer

❖ What are the values of fundus examination:

1) Hypertensive retinopathy	2) Diabetic retinopathy
3) Infective endocarditis : ▪ In central Retinal artery : occlusion by embolism gives snow white Retina ▪ Roth spots (vasculitis): small red petichae with pale white centers	4) Systemic lupus erythematosus : ▪ Hard exudates (cytoid bodies) ▪ Hemorrhages
	5) Arterial pulsation in AR

❖ What do you know about Xanthomas (Xanthomata) ?

Definition : **Xanthomas** are smooth surfaced **yellow** or **orange** plaques or **nodules** in the skin due to focal dermal aggregations of lipid-loaded cells (foamy histocytes)

Different types of hyperlipidemia may induce varying patterns of xanthomas:

1. Xanthelasma : Subcutaneous deposits of cholesterol just medial to the eyelids (with type IIA & III or without lipid abnormality)	2. Plane xanthoma: - On palmer creases (Type III) - Also occur with primary biliary cirrhosis
3. Tuberous xanthoma: Over the joints especially knees and elbow (type IIA, III)	4. Tendinous xanthoma: Over the tendons e.g. Achilles tendon , patellar tendon & finger extensor tendons (Types IIA & III)
5. Eruptive xanthoma: - Occur on the buttocks, posterior thighs (hyperlipidemia type I , IIB, III, IV & V) - Also occur with primary biliary cirrhosis	
Treatment : Lipid lowering agents, Plasmapheresis & Liver transplantation	

1. Xanthelasma :



2. Plane xanthoma:



3. Tuberous xanthoma:



4. Tendinous xanthoma:



5. Eruptive xanthoma:



FREQUENTLY ASKED QUESTIONS FOR MID & END ROUND EXAM

Test yourself!

1. What is meant by body mass index?	2. What are the causes of orthopneic position ?
3. What are other decubiti you know ?	4. What is normal heart rate?
5. What are the causes of bradycardia?	6. What are the causes of tachycardia?
7. What is meant by pulse deficit?	8. How to differentiate between irregular pulse of AF & extrasystoles?
9. What are the causes of big pulse volume?	10. What are the causes of small pulse volume ?
11. What are the causes of variable pulse volume?	12. What is meant by pulsus alternans?
13. How to diagnose pulsus alternans by sphygmomanometer?	14. What is the mechanism of pulsus alternans in severe LVF?
15. What is meant by pulsus paradoxicus?	16. How to diagnose pulsus paradoxicus by sphygmomanometer?
17. What are abnormal pulse characters ?	18. What are the synonym of water hammer pulse?
19. What are the causes of unequal pulse volume?	20. What is meant by radiofemoral delay?
21. What is mean by force & tension ?	22. Why do we use palpatory method before auscultatory method in measurement of ABP?
23. What is the auscultatory gap ?	24. What is normal BP?
25. What is normal temperature?	26. What is the difference between fever & hyperpyrexia?
27. What are causes of puffiness of eye lids?	28. What are causes of red glazed tongue?
29. What are the causes of bad odour of the mouth?	30. Define jaundice?
31. What is the normal level of serum bilirubin?	32. Why do you inspect jaundice in daylight?
33. Why do you inspect jaundice in sclera?	34. What are other causes of yellow skin? = what is the DD of jaundice ?
35. What is the difference between different types of jaundice?	36. Define cyanosis ?
37. What are the causes of central cyanosis ?	38. What are the causes of peripheral cyanosis ?
39. What produced combined central & peripheral cyanosis ?	40. How to unmask peripheral cyanosis in a patient with combined central & peripheral cyanosis ?
41. Can severe anemia produce central cyanosis?	42. What is meant by differential cyanosis?
43. Why doesn't peripheral cyanosis affect the tongue ?	44. When can peripheral cyanosis affect the tongue?
45. What is the difference between central & peripheral cyanosis?	46. What are the causes of butterfly rash?
47. Where would you like to see pallor ?	48. What are the causes of pallor?
49. What is the normal JVP?	50. What are the causes of ↑ JVP (congested neck veins)?
51. What are the causes of systolic expansion of neck veins?	52. What are causes of giant a wave ?
53. Which is better to examine IJV or EJV & why?	54. Which is better to examine Rt IJV or Lt IJV?
55. What are the causes of congested non pulsating neck veins?	56. What are the differences between neck vein & carotid pulsations?
57. What are the causes of carotid shudder?	58. What are the causes of spider nevi?
59. What is the distribution of spider nevi?	60. What is the mechanism of spider nevi?
61. Differential diagnosis of spider nevi?	62. What are the causes of tender edema of LL?
63. What are the causes of bilateral edema?	64. What is the mechanism of clubbing?
65. What are types of clubbing?	66. What are the degrees of clubbing?
67. What is the test that detects mild clubbing?	68. What are the cardiac causes of clubbing?
69. What are the chest causes of clubbing?	70. What are the abdominal causes of clubbing?
71. What are the causes of palmer erythema ?	72. What are the causes of flapping tremors ?
73. What can cause spooning of nail ?	74. What are the causes of white nail?
75. What are the causes of Dupetryn's contracture?	76. What are the special characters with carotid artery & aortic valve disease ?
77. What are the indications to measure BP in lower limbs?	78. Causes of orthopnea?
79. All questions answered in the clinical discussion are very important	

SUMMARY OF GENERAL EXAMINATION

I. Vital signs

1. **Temperature:** orally : Normal : 36.5 – 37.2 °C

2. **Pulse : RR-VV-SEP**

- Rate : normally : 60-100 b/m
- Rhythm : regular or irregular (AF)
- Volume : average , small or big.
- Condition of vessel wall: not felt
- Equality on both side in pulse volume: equal
- Peripheral pulsations: inatet

3. **Blood pressure**

• Normal range:

Systolic from 90 to 140 mmHg
Diastolic from 60 to 90 mmHg

4. **Respiratory movements :**

- Rate : Normal : 12-20/min
- Rhythm : regular
- Depth : average
- Type of breathing :
 - Females : thoraco-abdominal
 - Males : abdmno-thoracic

II. General overview; ABCCDE

❖ **Appearance, Built & height, Consciousness, Colours, Decubitus & facial Expression.**

5. **Colour:**

1. **Jaundice:** Examined in lower fornix of the sclera ; asking the patient to look upwards and facing the day light
2. **Cyanosis:** in undersurface of tongue & hands
3. **Pallor:** slightly pull the lower lip outward & look for the colour inside, palmer creases

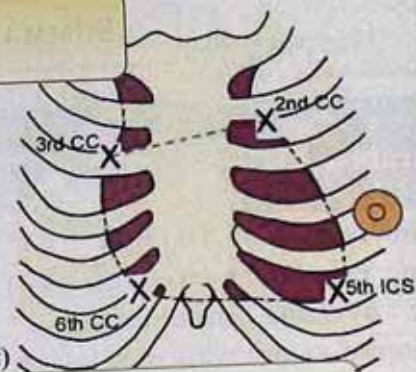
III. Systemic overview

A. Head	B. Neck	C. Upper limbs	D. Lower limbs
1. Head 2. Eye 3. Nose 4. Butterfly area 5. The mouth: ▪ Angle of the mouth ▪ Lips ▪ Teeth ▪ Tongue: - Size - Colour - Papillae - Surface - Movements ▪ Buccal mucosa ▪ Palate ▪ Uvula ▪ odour 6. Parotid gland	1. Skin 2. Lymph node 3. Pulsations: • Carotid pulsations • Neck veins 4. Trachea 5. Thyroid	1. Skin of the hand 2. Shape of the hand 3. LN of the hand 4. Pulse & BP 5. Palm 6. Nail: TCKSLY 7. Nodules 8. Clubbing 9. Colours 10. Trophic changes 11. Tremors: • Fine tremors • Coarse "flapping" tremors 12. Joints & Deformity 13. Muscles & nerves	• Oedema • As upper limb
			E. Other systems except that of local examination

CARDIOLOGY

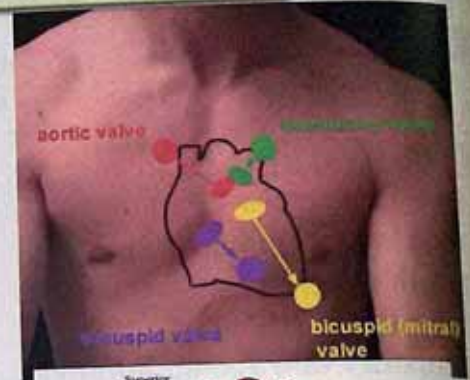
❖ Surface anatomy of the heart : 4 points

- Right border : represented by a line between two points:
 - Right 3rd costal cartilage 3 cm from midline
 - Right 6th costal cartilage 3 cm from midline
- Left border : represented by a line between two points
 - Left 2nd costal cartilage 4 cm from midline
 - Left 5th intercostal space 9 cm from midline (apex of the heart)

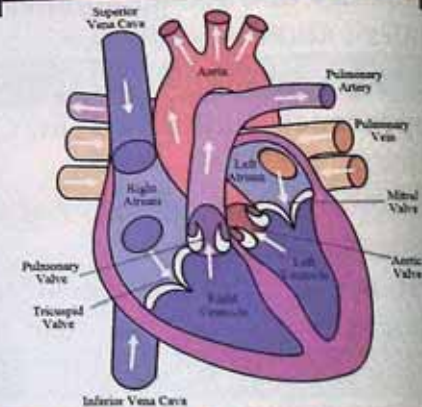
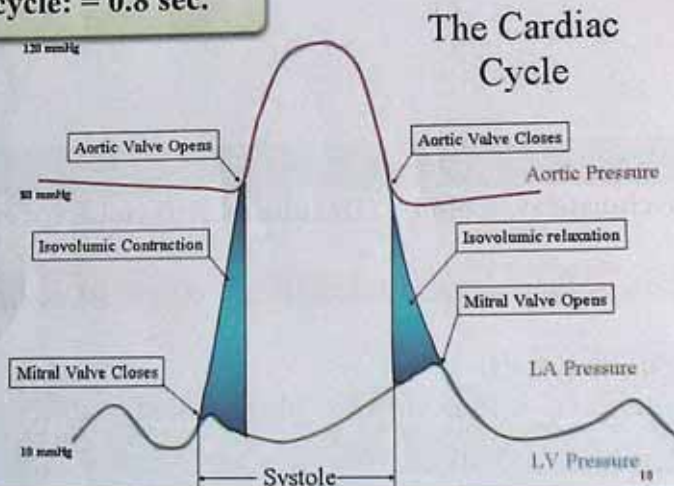


❖ Surface anatomy of cardiac valves : adjacent to left border of sternal body :

- The SemiLunar (SL) valves :
 - Pulmonary valve : left 3rd sternocostal junction
 - Aortic valve : left 3rd intercostal space (lateral border of sternum)
- The AtrioVentricular (AV) valves :
 - Mitral valve : left 4th sternocostal junction
 - Tricuspid valve : left 4th intercostal space (behind center of sternum)



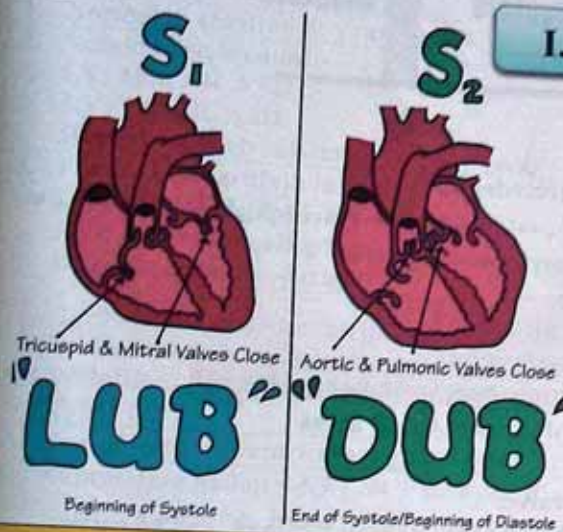
❖ Cardiac cycle: = 0.8 sec.



I. Systole (0.3 sec) :	II. Diastole (0.5 sec) :
1. First heart sound :	4. Second heart sound:
The cardiac cycle start by contraction of ventricles resulting in closure of mitral & tricuspid valves producing the first heart sound (S1)	After evacuation of blood, the ventricles relax, so the pressure in the aorta and pulmonary artery will exceed the pressure in the ventricles resulting in closure of aortic & pulmonary valves producing the second heart sound (S2)
2. Isometric contraction phase:	5. Isometric relaxation phase:
The ventricles continue to contract while the 4 valves of the heart are closed, so the pressure inside the ventricles rises rapidly without change in the volume	The ventricles continue to relax while 4 valves of the heart are closed, so pressure inside the ventricles falls rapidly without change in the volume.
3. Ejection phase:	6. Ventricular filling phase:
- When the pressure in the ventricles exceeds the pressure in the aorta & pulmonary artery, the aortic & pulmonary valves will open (normally with no sound) - Then the blood rushes from the ventricles to the aorta and pulmonary artery, - First rapidly: maximum ejection phase - Then slowly: reduced ejection phase	- When the pressure in the ventricles becomes lower than the pressure in the atria, blood flows to the ventricles passively, - First rapidly : maximum filling phase - Then slowly : reduced filling phase
8. Ventricular contraction: occurs again & the cycle is repeated.	7. Atrial systole:
- N.B.: any change in heart rate , is a change in diastole, systole is constant : - Tachycardia shortens diastole while Bradycardia lengthens diastole	The last amount of blood in the atria is pushed actively by atrial contraction in the late diastole.

❖ Heart Sounds and their relation to cardiac cycle:

- I. Heart sounds (S₁, S₂, S₃, S₄)
- II. Additional heart sounds (Systolic clicks & opening snap)
- III. Murmurs
- IV. Others heart sounds (Pericardial rub, pericardial knock, crepitations & metallic click)

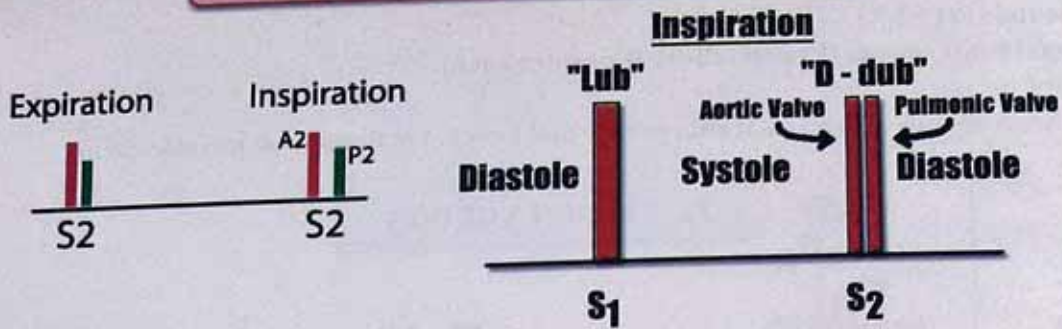


I. Heart sounds



1. First heart sound, S ₁	2. Second heart sound, S ₂
❖ Time:	❖ Time:
▪ Beginning of systole	▪ Beginning of diastole
❖ Caused by :	❖ Caused by :
▪ Valvular: closure of A-V (mitral & tricuspid) valves ▪ Muscular: early ventricular contraction vibrations of chorda tendinae & papillary muscles producing transient vibrations	▪ Valvular: Closure of semilunar (aortic & pulmonary) valves ○ Over aortic area : S₂ is single (A₂ only heard) ○ Over pulmonary area : S₂ is split (A₂ followed by P₂)
❖ Site of maximum intensity :	❖ Site of maximum intensity :
▪ At the apex of the heart	▪ Over base of the heart
❖ Intensity:	❖ Intensity:
☞ Causes of Accentuated S ₁ :	☞ Causes of Accentuated S ₂ :
1. MS - TS	1. Hyperdynamic circulation
2. Hyperdynamic circulation	2. ↑ A ₂ in systemic hypertension
3. Systemic hypertension	3. ↑ P ₂ in pulmonary hypertension
4. Short P-R interval (tachycardia nodal rhythm, Wolff-Parkinson-White syndrome (WPW))	4. Children & thin chest
5. Children & thin chest	☞ Causes of Weak (muffled) S ₂ :
☞ Causes of Weak (muffled) S ₁ :	1. ↓ A ₂ in AS & AR
1. MR - TR - calcified MS	2. ↓ P ₂ in PS & PR
2. Impaired myocardial contractility (HF, Myocardial infarction, myocarditis)	3. Hypotension
3. Hypotension	4. Mechanical: (causes of absent apex; see later)
4. Long P-R interval (bradycardia, heart block)	❖ N.B.:
5. Mechanical (causes of absent apex; see later)	➤ Aortic component of S₂ is heard all over heart (including apex) while Pulmonary component is heard over Pulmonary area only. ➤ In Pulmonary hypertension S₂ is closely split ➤ Splitting of S₂ : see later.
➤ Causes of Variable S₁ intensity : due to variable P-R interval in: ○ AF ○ Extrasystoles ○ Complete heart block ➤ Cause of Splitting of S₁ : right bundle branch block	

❖ Splitting of Second heart sound :



- Normally : LV ejection period is shorter than that of RV , So A2 precedes P2
هتسبح السماعه على ال P.area هتسمع صوت واحد , قول للعيان ياخذ نفس , هتلاقي الصوت اتقسم صوتين
- Timing of A2 in relation to P2 : depends on period of ejection of corresponding vessel

❖ Relation of splitting to respiration:

⚠ Splitting Is Only Detected Over Pulmonary Area as pulmonary component is weak while aortic component is loud heard at both A & P areas

- During inspiration :
 - ↑ VR will increase RV Load , with delayed closure of pulmonary valve.
 - Meanwhile the lung is expanded & retains more blood in its vessels with decrease in LV load, and so earlier closure of Aortic valve
- So during inspiration → wide splitting of S2
- During expiration: the reverse occurs → narrow splitting or fusion (single S2)

? What are the causes of :

❖ Wide splitting Of S2: "delay of pulmonary closure"	❖ Reversed "paradoxical" splitting: "delay of aortic closure"	❖ Single S2
هتسبح السماعه على ال P.area هتسمع صوتين , قول للعيان ياخذ نفس , هتلاقي الصوتين يبعثوا عن بعض أكثر	هتسبح السماعه على ال P.area هتسمع صوت صوتين , قول للعيان ياخذ نفس , هتلاقي الصوتين بقوا صوت واحد	هتسبح السماعه على ال P.area هتسمع صوت واحد , قول للعيان ياخذ نفس , هتسمع صوت واحد
- Delayed P2 in PS - RBBB - VSD - ASD	- Delayed A2 in AS - LBBB - PDA	- Synchronous A2 & P2 : single ventricle defect - Faint A2 (P2 only heard): AS - Faint P2 (A2 only heard): PS (alone or with F4)
Causes of wide fixed splitting of S2 : "unaffected by respiration" → ASD هتسبح السماعه على ال P.area هتسمع صوتين , قول للعيان ياخذ نفس , هتلاقي الصوتين زي ما هم لا يحدث اي تغير	Reversed splitting due to: Delay of A2 to come after P2 → reversal of relation to respiration, i.e. Splitting closes during inspiration & widens during expiration	Closed narrow split: Pulmonary hypertension

3. Third heart sounds, S3

- Early diastole sound heard shortly after S2 best heard by the cone in left lateral position.
- Excess ventricular vibrations (blood gush in ventricle after opening of A-V valve)

Time:

- Late diastole (presystolic) sound heard just before S1 best heard by the cone in left lateral position.

Mechanism:

- Forceful atrial contraction against high ventricular end diastolic pressure

Causes of the sound:

1. Normal in children & young adult
2. Volume overload in:
 - LV: MR, AR, VSD
 - RV: TR, PR, ASD
 - Hyperkinetic circulation
3. Flabby myocardium in:
 - LVF (apical gallop)
 - RVF (tricuspid gallop)
 - Dilated cardiomyopathy

1. Normal in elderly person
2. Pressure overload in:
 - LV: systemic hypertension, AS
 - RV: pulmonary hypertension, PS
3. Reduced compliance of ventricles in:
 - Myocardial infarction
 - Hypertrophic cardiomyopathy

❖ Gallop rhythm "triple rhythm" صوت جري الحصان

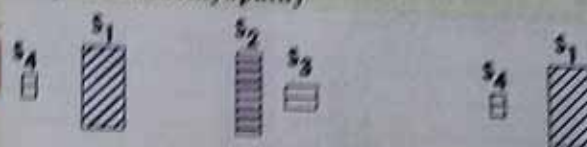
- ❖ Definition: gallop = (S3 or S4 or Both) + tachycardia
- ❖ Types :

A. Protodiastolic (ventricular) gallop = S3 + tachycardia

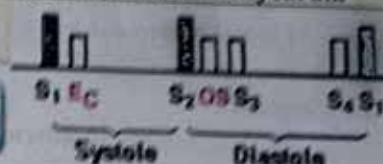
C. Summation gallop = S3 + S4 + tachycardia

- Cause e.g. hypertensive heart failure

B. Presystolic (atrial) gallop = S4 + tachycardia



II. Additional Heart sounds



A. Systolic clicks

1. Early systolic = ejection click (E.C.)

2. Mid & late systolic = mitral click

B. Opening snap

❖ Mechanism

- Valvular E.C.: Opening of stenosed semilunar valve
- Vascular E.C.: Dilatation of the corresponding vessel

- Upward prolapse of mitral leaflets into left atrium during systole.

- Opening of stenosed A-V valves. (stenosed valve with fibrosed rigid periphery and pliable center)

❖ Identification:

❖ Time:

- Early systolic sound heard shortly after S1

- Mid to late systolic & it's timing change with posture

- Diastolic sound heard closely after S2 (separated from it by isometric relaxation phase)

❖ Site of maximum intensity :

- Aortic & pulmonary areas (base of the heart)

- Apex

- Midway between apex & left sternal border

❖ Character :

- Sharp, high pitched, clicky sound

- Sharp, high pitched, clicky sound

- Sharp, high pitched, snappy sound

❖ Causes:

- Aortic ejection click in:
 - Valvular AS
 - Systemic hypertension with aortic dilatation

- Pulmonary ejection click in:
 - Valvular PS
 - Pulmonary hypertension with pulmonary dilatation

- Mitral valve prolapse "click murmur syndrome"

- Organic MS or TS

❖ Significance :

- Diagnostic of organic MS
- Denotes mobile non-calcific leaflets with no regurge
- Assess the Severity of MS in inversely proportional to S2 - opening snap interval i.e. short interval → severe stenosis

❖ Significance :

- The stenosis is valvular (never sub or supra valvular)
- The valve is non calcific.

Mid-systolic Click



III. Murmurs لفظ

❖ **Definition** : musical sounds produced by turbulent blood flow

❖ **Mechanism**:

1. Flow into a dilated chamber e.g. pulmonary dilatation
2. Flow across a septal defect or a shunt
3. Flow across a stenosed valve
4. Regurgitant flow across an incompetent valve
5. Increase amount or rate of blood flow; functional murmur

❖ **Synonyms**:

Over the heart :murmur Over arteries : bruit Over veins : hum

❖ **Types of murmur**:

1. Organic murmur	2. Functional = relative = flow murmur
Caused by structure anatomical abnormality in heart or vessel	Caused by functional (physiological) abnormality that increases the blood flow through a structurally normal heart i.e. relative stenosis
Usually <u>loud</u>	Usually faint
Usually <u>long</u>	Usually <u>short</u>
Usually harsh	Usually <u>soft</u>
May propagate to other areas	Localized
Associated with thrill + symptoms & signs	No thrill - no symptoms nor signs
Medical treatment has no effect	May disappear after treatment e.g. anemia

3. **Innocent** :

- Turbulence in absence of structural or physiological abnormalities (i.e. No lesions at all)
- It occurs in children & disappears as they grow

❖ In describing murmur , we comment upon the following items : SACT.R

1. Site of maximum intensity & Area of propagation
2. Character
3. Timing
4. Thrill & Grade
5. Type
6. Relation to position, respiration, exercise

1. Site of maximum intensity & Area of propagation

❖ Lesion	❖ Site of maximum intensity	❖ Area of propagation
MS	Mitral area; Apex	Localized
TS	Tricuspid area	Localized
MR	Mitral area; Apex	- If anterior leaflet : Axilla - If posterior leaflet : back (left scapular region) & sternum
TR	Tricuspid area	Apex
AS	1 st aortic area	Apex & carotids
PS	Pulmonary area	Tricuspid area & may be under left clavicle
AR	2 nd aortic area	Apex
PR	Pulmonary area	Tricuspid Area
VSD	Left parasternal area	Whole precordium
PDA	Left infraclavicular area	Pulmonary area
Aortic coarctation	Inter scapular area	Anteriorly

2. Character:

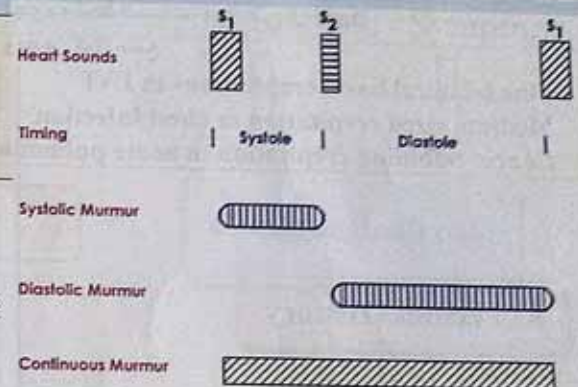
- Soft : all incompetent murmurs & flow murmurs
- Harsh: VSD, COA & all stenotic murmurs except MS.
- Machinery: PDA
- Rumbling: MS & TS
- Musical "Sea-gull murmur": due to rupture of valve complex (cusps, chordae & papillary muscle), mostly due to endocarditis or ischemia.

3. Timing: timed against carotid pulse

A. Systolic :				B. Diastolic :	
Early systolic	Ejection systolic	Late systolic	Pan or holosystolic	Early diastolic	Mid & late diastolic
Very small VSD	- AS - PS	- Mitral valve prolapse (MVP) - Hypertrophic obstructive cardiomyopathy	- MR - TR - VSD	- AR - PR: (Graham Steell murmur)	- MS & TS - Carey Coomb's murmur: (functional MS in RF)

C. Continuous; systolic & diastolic murmur :

- Venous hum : jugular vein hum in the neck
- Arterio-venous fistula : systemic, pulmonary & coronary
- Patent Ductus Arteriosus (loudest in 2nd left interspace)
- Double valve lesion : AS/AR - MS/MR
- After Blalock-Taussig operation



4. Thrill & grade :

- ❖ Thrill is Palpable loud murmurs like sensation of purring cat
- ❖ Grade 4/6 at least.
- ❖ Due to organic & not functional valve disease.

Stenotic murmurs are harsh + marked thrill	Regurge murmurs are soft + minimal thrill	Functional murmur : no thrill
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♥ Grades (intensity) of the murmur: Levine grading system e.g. Grade .../VI

- Grade I : very faint- heard by an expert in optimum condition (quiet room-proper patient position)
- Grade II : faint - heard by non-expert in optimum condition
- Grade III : moderate intensity - easily heard but no thrill
- Grade IV : loud with thrill يتسمع والسماعة لازقه كلها
- Grade V : very loud - heard over wide area , with thrill يتسمع بحرف السماعة
- Grade VI : extremely loud with thrill - be heard without stethoscope. يتسمع من بعيد

5. Type : organic or functional murmur (see before)

6. Relation to:

A. Posture "Position"	B. Respiration	C. Exercise
Mitral murmurs : best heard in left lateral position	- Right (tricuspid & pulmonary) Sided murmurs : ↑ by inspiration (Carvallo's sign) (due to ↑ VR → ↑ blood flow) → Carvallo's sign: differentiate between TR & MR	↑ MS, MR. & AR

♥ N.B.: Length of the murmur:

- Length of the murmur is related to the severity of the underlying lesion
- Organic murmurs are long while functional murmurs are short
- The loudness of the murmur is not related to the severity of the lesion

IV. Other heart sounds

1. Pericardial rub	2. Pericardial knock
▪ Cause : Pericarditis	▪ Cause : Constrictive pericarditis
❖ Character	
• Superficial leathery continuous sound • Site: Best heard at left sternal line over bare area & patient is sitting & leaning forward. • تقعد العيان , وتخلي العيان يميل لقدام وسماعتك على ال Left sternal border • دوس جامد على السماعه وقول للعيان وقف نفس Pericardial rub Increased by pressing stethoscope against chest wall • دوس جامد : Pericardial rub does not disappear on holding up respiration	• Heard at same time of S3 • Sudden halting of relaxing ventricles by rigid pericardium
3. Crepitations :	4. Metallic click :
• تقعد العيان على السرير , يرفع الهدوم وتحط السماعه ورا على ال Base • تقول للعيان انهج وبعدها كحح , وبعدين كمل سمع • Fine bilateral basal crepitations in LVF • Medium sized crepitation in chest infection • Coarse bubbling crepitation in acute pulmonary edema	• Metal prosthesis = Metallic sound

CARDIAC HISTORY

A. Personal History

1. <u>Name</u> : الإسم ثلاثي	2. <u>Age</u> : ○ Young : congenital heart disease ○ Middle to old : coronary congenital heart disease	3. <u>Sex</u> : ○ Females : MS, rheumatic chorea ○ Males : MR, coronary heart disease
4. <u>Occupation</u> : ○ Stressful job : coronary heart disease, hypertension	5. <u>Marriage</u> : recent delivery for DVT & pulmonary embolism 6. <u>Menstrual history</u> if female.	7. <u>Residence & address</u> : rheumatic fever is common in rural areas & low social level
8. <u>Habits of medical importance</u> : 1. Cigarette smoking : coronary heart disease, arrhythmia 2. Alcohol : alcoholic cardiomyopathy 3. IV addicts : fungal & staph acute bacterial endocarditis		9. <u>Handedness</u> .

B. Complaint + duration: ؟ إي اللي جابك المستشفى

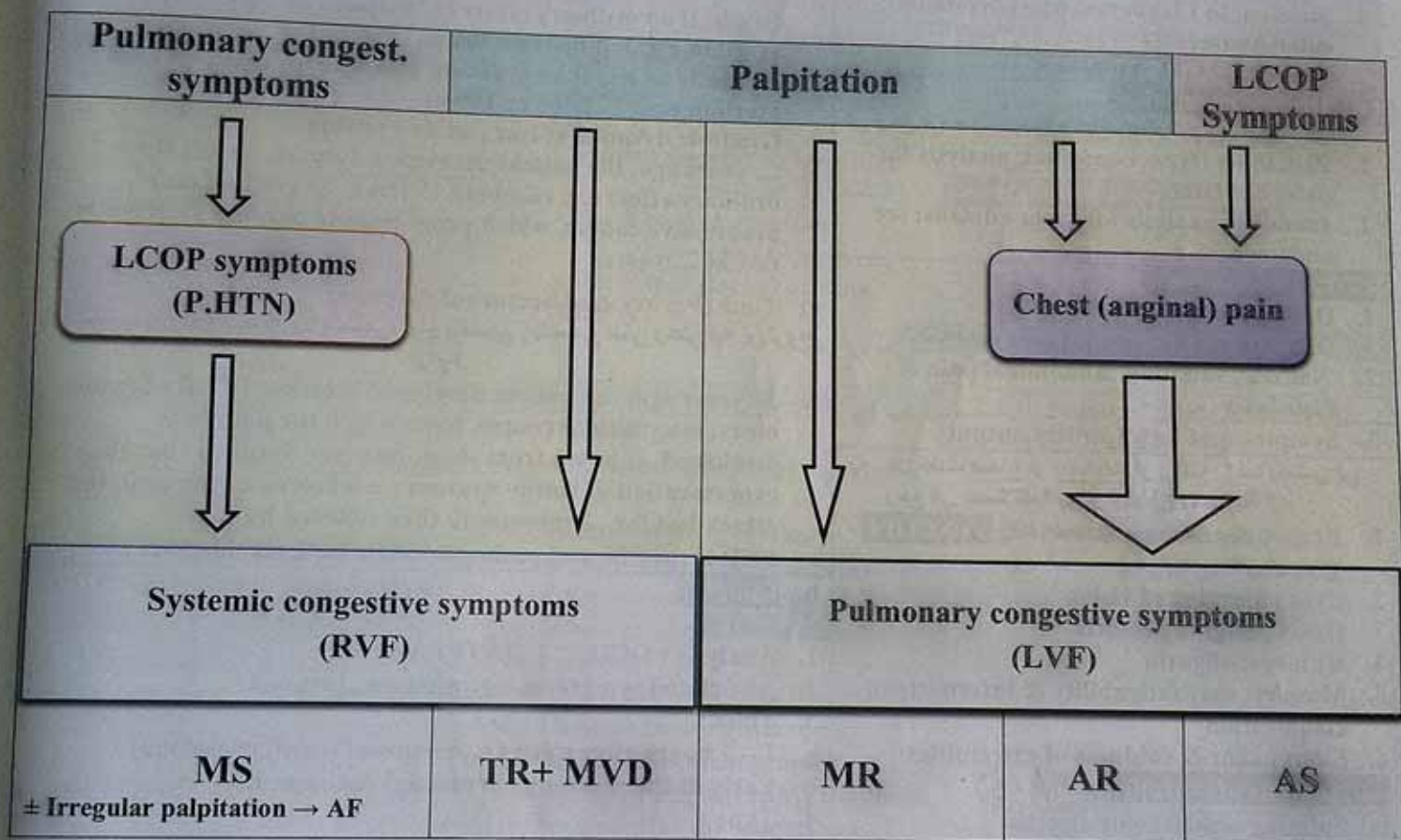
❖ Common complaints :

▪ Shortness of breath.	▪ Awareness of heart beats.	▪ Chest pain, fainting , easy fatigability	▪ Lower limb swelling.
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C. History of present illness = HPI ؟ المرض بدأ معاك إزاي , آخر مرة كنت كويس فيها إمتى ؟

1. Symptoms of systemic Congestion indicating right side heart failure (TR)	2. Symptoms of Pulmonary Congestion indicating left side heart failure (Mitral Valve Disease = MVD)
3. Symptoms of Low Cardiac out put	4. Symptoms of Congenital heart disease
5. Chest Pain (Ischemic pain; Angina)	6. Pressure symptoms
7. Palpitation	8. Pyrexia (fever)
9. Symptoms of increased Blood pressure (Hypertension)	10. Symptoms of occlusion of Blood vessels (systemic embolization)
11. Investigations & treatment received	12. Symptoms of other systems

Algorithm of Cardiac History For Diagnosis



❖ N.B.:

- Double mitral : regular palpitation on top of pulmonary congestion symptoms
- Irregular palpitation → AF → MVD (MS > MR) or double mitral lesion.

❖ N.B.:

تكتب في ال complaint بكلام العيان
تكتب في H/O of present illness عادي

1. Symptoms of systemic Congestion indicating right side heart failure رجلك انتفخت او ورمت ؟ A. Lower limb edema ؟ LL swelling: analysis: 1. Onset, Course, Duration (OCD), what increase (↑), what decrease (↓) 2. Relation to Ascites : onset before or after LL edema 3. Pitting or no? لا؟ الشراب بيعلم ولا؟ 4. Painless or painful لما بتكوس على الورم بيوجعك ولا لا؟ 5. Laterality (Unilateral or Bilateral) 6. Level (extent) A. Ascites ؟ Abdominal distension : 1. Relation to LL edema: onset before or after Ascites 2. Associated GIT symptoms B. Hepatic congestion: في وجع في جنبك اليمين وعينك اصفرت ولون البول اتغير ؟ 1. Pain in Rt Hypochondrium: analysis for pain: See later. 2. Jaundice; analysis : for more details; see abdomen C. GIT congestion : 1. Dyspepsia ؟ بينعبك ؟ - Discomfort after meals ما بترحينش 2. Nausea , vomiting, Abdominal pain & Flatulence 3. Symptoms of Low Cardiac output هل عندك صداع او اغماءات او زغلله ؟ لما بتعشي في وجع في عضلاتك ؟ بهتان والاطراف ساقعة ؟ 1. Brain : headache, dizziness & SYNCOPE بيغشي علي او بدوخ 2. Eyes : blurring of vision 3. Heart : angina pectoris 4. Kidneys: oliguria 5. Muscles: easy fatigability & intermittent claudication 6. Skin: pallor & coldness of extremities Analysis of syncope : A.S Fainting or black out attacks ○ OCD, ↑, ↓. ○ Associated symptoms (pre or post) هل معاها : - Palpitation (analyze), بتيقي حاسس بضربات قلبك - أو حاسس برفرقة على صدرك؟ - Chest pain (analyze), وجع في صدرك - Cold extremities جسمك بيسقع - Sweating عرق , etc.... ○ Type of syncope مع المجهود ولا بدون مجهود : at rest or <u>exertion</u> (during or after exercise) ○ Time of the attack بتستمر اذ اي ○ Recovery : <u>spontaneous</u> or with intervention بتفوق لوحده ولا لازم حد يفوقك ○ Frequency of attack جتلك كام مرة ○ With <u>convulsion</u> (to exclude neurological cases) or not? اتشنجت ؟ ○ With <u>cyanosis</u> (to exclude F4) or not? ازرقيت ؟	2. Symptoms of Pulmonary Congestion indicating left side heart failure Dyspnea ؟ هل عندك كرشة نفس , نهجان ؟ (shortness of breath); analysis: 1. OCD. ↑, ↓. 2. Associated symptoms e.g. palpitation, chest pain & wheezes هل مع كرشة النفس فيه وجع او رفرقة او صدرك بيزيق ؟ 3. <u>Relation to :</u> a) Position (orthopnea; using extra pillows) في وضع معين بتنام فيه ؟ بتنام على كام مخدة ؟ The patient had orthopnea; he must sleep on ... pillows. b) Exertion (on effort) & it's grade (usually grade 3) : بتحصل مع المجهود زي طلوع السلم أو الشغل أو الجري ؟ Grade 1: Not more than ordinary effort i.e. dyspnea on severe exertion e.g. 3rd floor or 300 m. Grade 2: on ordinary effort i.e. dyspnea on moderate exertion e.g. 2nd floor or 200 m. Grade 3: on less than ordinary effort i.e. dyspnea on mild exertion e.g.: 1st floor or 100 m. Grade 4: dyspnea at rest , on no exertion ... years ago, the patient developed dyspnea on less than ordinary effort e.g. climbing 1st floor , of gradual onset, progressive course, which progressed to become dyspnea at rest in ... years. c) Time (Paroxysmal nocturnal dyspnea) ما بتنام بتكمل نوم للصبح ولا بتصحى ؟ ولو بتصحى , بتصحى امتى وعلى ايه , وبتروح ازاى ؟ ... years ago, the patient developed attacks of PND of acute onset, intermittent course, from which the patient is awakened ... hours from sleep, on sever dyspnea , cough , expectoration of frothy sputum (± wheezes ± cyanosis), the attack last for ... minutes & then relieved by • PND is specific and pathognomonic for Left sided HF. b. Cough : ○ Cough 1. Analysis : OCD, ↑, ↓, (ATP) 2. Associated symptoms e.g. wheezes , toxemia 3. <u>Time</u> : ○ 1 – 2 hours after sleep i.e. <u>nocturnal</u> (cardiac asthma) ○ Early in the morning (Bronchial Asthma; BA) 4. <u>Type</u> : ○ Continuous هل الكحة ديه مستمرة طول الوقت ؟ ○ Paroxysmal ولا في صورة نوبات : بتيجي وتروح : متقطعة ؟ 5. Relation to <u>Position</u> & exertion (cardiac cough: ↑ on lying flat & exertional) هل الكحة بتزيد مع المجهود ولا لا ؟ في وضع معين بيزود الكحة ؟ هل الكحة بيلغم ولا كحة ناشفة ؟ 6. <u>Dry</u> (but maybe whitish sputum) or <u>Productive</u> (Expectoration) ناشفة or بيلغم ; If productive; analyze (see chest). Hemoptysis ؟ هل بتكح دم او بتكح بلغم معرق بدم ؟ (Coughing of blood) : For more details about analysis see chest. ▪ OCD, ↑, ↓ ▪ A: Attack : number, date, before the attack..., during the attack..., after the attack ... ▪ B: Blood : amount, content, color ▪ C: Circulatory compensation : hospital admission, blood transfusion ▪ D: Dysfunction : Disturbed conscious level, bleeding from other orifices Recurrent Chest infection ؟ في التهابات على الصدر بشكل متكرر ؟ ○ Fever, productive cough with (yellowish or greenish) sputum possible culture & sensitivity & antibiotic use.
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4. Symptoms of Congenital heart disease A. Cyanotic group: شفايفك بتزرق - ضوافرك بتزرق ؟ ○ Age of onset: • Since birth : TGA • Shortly after birth : F4 • Few years (3-10) after birth : F3 • In teenager : Eisenminger's ○ Type : central, peripheral, differential ○ Cyanotic spells relieved by squatting in F4 ○ Associated congenital anomaly e.g. polydactyl B. Acyanotic group: winter bronchitis & exertional cyanosis in Left to Right shunt ❖ IF -VE : No symptoms suggestive of congenital HD (late onset of complaint, no cyanosis, -ve consanguinity)		
6. Chest Pain هل عندك وجع في صدرك ❖ Usually in Aortic valve disease & ischemic heart diseases; anginal pain ❖ <u>Analysis of any Pain at any site as following:</u> • OCD, ↑, ↓, Associated symptoms • Character of the pain: e.g. compression, burning, heaviness, stitching, ثقل, نغز أو كششة • Site of the pain: مكان الوجع ده فين • Radiation: هل يبسمع في حته تاني • Relation to meal, respiration, exertion, postural: هل له علاقة ب (meal, respiration, exertion, postural) ❖ IF -VE : no chest pain detected by the patient	7. Palpitation رفرفة, هل حاسس بضربات قلبك ؟ (awareness of heart beats) ❖ Usually in AR or MR cases ❖ <u>Analysis :</u> • OCD, ↑, ↓ • <u>Rhythm:</u> Regular or irregular منتظمة ولا غير منتظمة • At <u>Rest</u> or with exertion بدون مجهود ولا مع المجهود • <u>Rate:</u> Rapid (tachy) or slow (brady) سريعة ولا بطيئة	5. Pressure symptoms: هل صوتك انتبح, هل عندك صعوبة في البلع ؟ • Dysphonia (Hoarseness of voice) : pressure on left recurrent laryngeal nerve • Dysphagia (difficulty in swallowing) : pressure on esophagus • Dyspnea & cough : pressure on tracheobronchial tree ❖ IF -VE : no symptoms suggestive of pressure manifestations e.g. Hoarseness of voice, dysphagia
10. Symptoms of occlusion of Blood vessels (systemic embolization) هل حصل ثقل في لسانك أو ايدك أو رجلك ؟ • Weakness : embolization of middle cerebellar artery • Chest pain : embolization of coronary artery • Bleeding in urine : embolization of renal artery • Pain in left hypochochondrium : embolization of splenic artery ❖ IF -VE : No symptoms suggestive of systemic embolization	9. Pyrexia (Fever)? هل سخنت • OC(type)D • Associated symptoms e.g. rigors رعشة • History of drug intake ❖ IF -VE : No recurrent fever detected by the patient 11. Investigation & treatment received عملت فحوصات اي ؟ واخنت علاج اي ؟ ❖ Cardiac Investigations e.g. ○ ECG, Echocardiography, coronary angiography, thallium. ❖ Non cardiac investigation e.g. ○ Blood sugar ○ Lipid profile ○ Blood cultures ❖ Treatment received e.g. ○ Digitalis, Diuretics ○ Anticoagulation ○ Long acting penicillin	8. Symptoms of increased Blood pressure: ➤ If patient knows : ○ The patient is not hypertensive ○ The patient is known to be hypertensive ... years on ... medication ➤ If patient does not know : Ask about Hypertension indirectly: في وش في ودانك وصداغ وزغلة ؟ ○ Headache, Blurring of vision, Tinnitus, Epistaxis
12. Symptoms of other systems e.g. chest, GIT, neurology هل عندك مشاكل تادية في جسمك ؟ وتأخذ كل سيستم وتسال العيان على الأعراض الكبيرة اللي فيه بسرعة أوي		

D. Past history as usual but focus on rheumatic fever

• Since ...	• Manifested by ...	• Investigated by ...
• Treatment ... & it's effect ...	• Continued or not	• Recurrence or not
• Recurrent tonsillitis, pharyngitis, fever, arthritis	• Intake of anti-inflammatory (aspirin & steroid)	• Tests for allergy with injection • IM injection of long acting penicillin; monthly.

E. Family history as usual & asked for congenital heart disease or rheumatic heart disease.

General Examination Of Cardiac Case

❖ Examination as general examination but stress on the following:

I. Vital signs

❖ Temperature	❖ Pulse	❖ Blood pressure	❖ Respiratory rate
See below	Regular or irregular (AF)	Hill's sign of AR	Tachypnea in LSHF
	Peripheral signs of AR in lower limbs		

❖ What are the Causes of fever in a cardiac case?

1. Endocardial	2. Myocardial	3. Pericardial	4. Pulmonary	5. Other
- Rheumatic fever or activity - SBE	- Acute Myocardial infarction - Myocarditis	Acute pericarditis	- Pulmonary infarction - Pulmonary infection	- DVT - Associated fevers

II. General overview

A. Appearance	B. Built	C. Consciousness	C. Colors	D. Decubitus	E. Expression
Looks ill & toxic in SBE	- Under built: cardiac cachexia - Over built: Pickwickian syndrome → cor-pulmonale	- DCL: in hypertensive encephalopathy - Cardiac causes of syncope e.g. AS	- Jaundice - Cyanosis - Pallor	- Orthopnea in severe HF - Prayer's position: pericardial effusion	According to the case e.g. Mongol in Down syndrome: VSD

❖ What are the Causes of ... in cardiac case ?

❖ Jaundice	❖ Cyanosis	❖ Pallor
- Associated: the commonest causes e.g. viral hepatitis - Hemolytic jaundice: in pulmonary infarction, hemolysis on artificial valves - Hepatocellular jaundice: in marked hepatic congestion (cardiac cirrhosis) due to severe right sided HF, pericardial effusion, TS & TR. - Obstructive jaundice: in marked hepatic congestion → compression of bile & inspissation of bile in canalicular lumen	- Central: in cyanotic congenital HD as F4, or advanced HF - Peripheral in low CO - Differential in PDA	- Anemia - Rheumatic activity - Infective endocarditis - Low cardiac output - shock

III. Systemic overview :

❖ Head	❖ Neck	❖ Upper limb	❖ LL
- Puffy eye lids in edema (Heart failure) - Nose & cheeks: malar flush of MS - De Musset's sign of AR - Conjunctiva: pallor & petechial hemorrhage in SBE	- Congested Neck veins: - Pulsating in congestive HF - Non pulsating in pericardial effusion - Carotid arteries: thrill in AR & AS - Peripheral signs of AR in the neck	- Nails: - clubbing: - Infective endocarditis (pale clubbing) - Congenital Cyanotic heart disease (cyanotic clubbing) - splinter hemorrhage in SBE - Nodules: - SC in Rh. fever - Osler's nodes in SBE - Peripheral signs of AR in UL	- Edema in HF, pericardial disease - Peripheral signs of AR in LL

IV. Other systems except that of local exam :

❖ Chest examination	❖ Abdominal examination	❖ Neurological examination
▪ As a cause : ○ Cor-pulmonale ▪ As a result : ○ Crepitations due to LVF & pulmonary oedema ○ Chest infection due to pulmonary congestion ○ Pleural effusion due to congestive heart failure ○ Lung collapse due to pericardial effusion (Ewart's sigs) ▪ As an association : ○ Dextrocardia in Kartagener's syndrome	▪ Liver: ○ Enlarged & tender in : RVF, pericardial disease ○ Enlarged, tender with expansile pulsation : TR, TS ○ Enlarged firm with sharp border : cardiac cirrhosis ▪ Spleen: ○ Enlarged in RVF, pericardial disease, infective endocarditis, portal hypertension in cardiac cirrhosis ▪ Ascites: ○ Ascites after edema in RVF ○ Ascites precox in TR, TS, pericardial diseases	▪ As a cause: ○ Autonomic neuropathy of the heart may cause silent myocardial infarction ▪ As a result : ○ Hemiplegia (embolic, thrombotic or hemorrhagic) ▪ As association : ○ Friedreich ataxia, Duchenne's myopathy, Rheumatic Chorea

Values of back examination in cardiac case

▪ Murmurs & Bruit: ○ MR : left scapular region ○ Coarctation of aorta : interscapular region ○ Bruit of renal artery stenosis in renal angle	▪ Crepitations: ○ Fine Bilateral basal crepitation : LVF ○ Medium sized crepitation in chest infection ○ Coarse Bubbling crepitations : acute pulmonary edema ▪ Ewart's sign : collapse of left lower lobe in pericardial effusion	▪ Vertebrae- Scapula : ○ Spina bifida : associated congenital anomaly in congenital HD ○ In ankylosing spondylitis: Low back pain, stiffness & limited spine mobility associated with AR ○ In coarctation of aorta: anastomosis around scapula in ▪ Skin: ○ Cafe' au lait patches in neurofibromatosis associated with pheochromocytoma & IHSS
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Local Examination Of Cardiac Case

Rules :

1. Good light
2. Exposure the patient's chest from umbilicus upwards.
3. Position of the patient supine with the head of the table slightly flexed
4. Always examine from the patient's right side
5. Make sure that the patient is comfortable in this position

Examination:

I. Combined inspection & palpation	II. Percussion	III. Auscultation
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I. Combined Inspection & palpation

1) Precordial bulge



- ❖ **Definition:** area of the chest overlying the heart (from the 2nd to 6th costal cartilage, from left sternal border to MCL)
 تعريف: المنطقة التي تغطي القلب: من عند حرف السريز وابطن Tangential مع ملاحظة مقارنة ناحيتي الصدر ببعض
- ❖ **Causes:** RV enlargement since early childhood due to Rheumatic HD or Congenital HD, & very rarely due to pericardial effusion

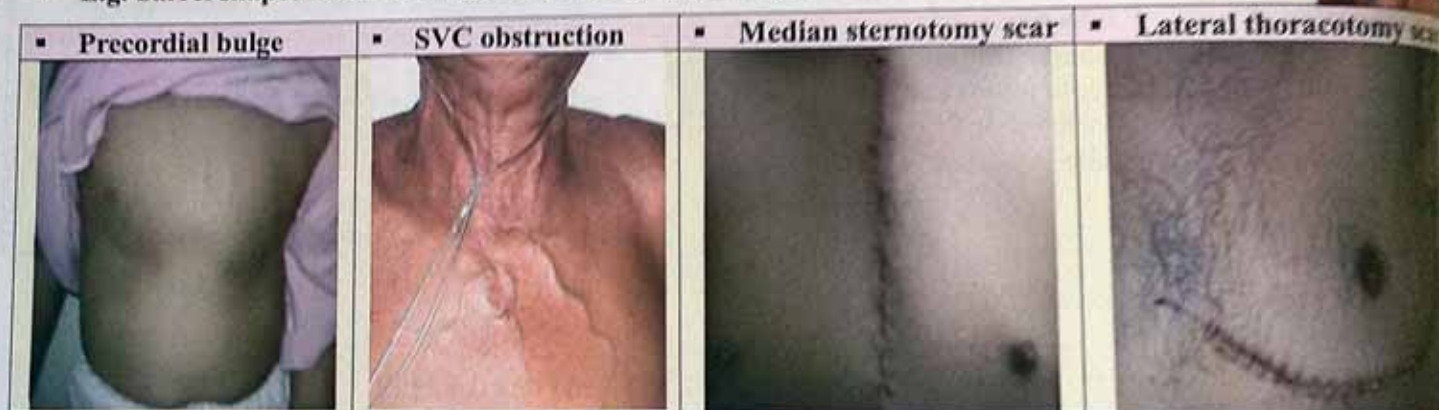
2) Veins & Scars

- ❖ **Dilated veins:** SVC obstruction (Filling upwards downwards)
- ❖ **Scars:** name, healing (1ry intention: clean linear healing, 2ry intention: pigmentation, keloid)
- Median sternotomy scar: open heart surgery e.g. valve replacement
- Lateral thoracotomy (inframammary) scar: closed heart surgery e.g. mitral valvotomy

3) Shape of the chest

Barrel shaped chest →

- E.g. barrel shaped chest: for more details see chest chapter



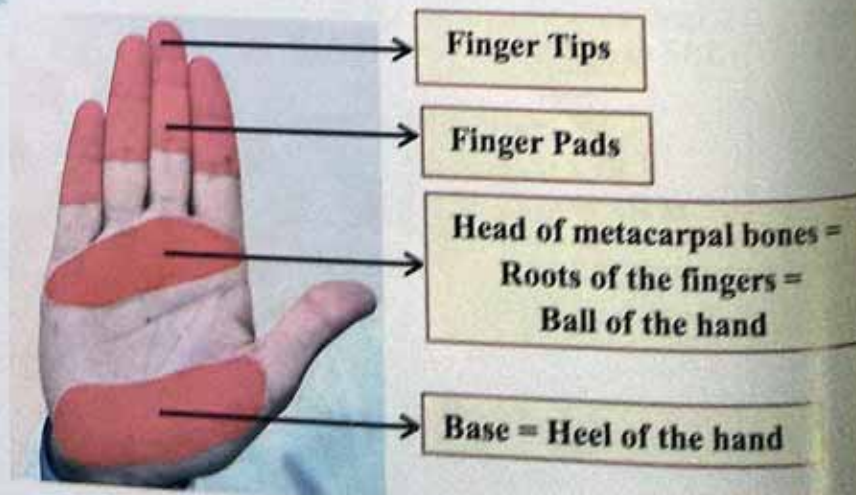
4) Pulsations لازم وقف نفسك في أي:

- 1) Apex
- 2) Suprasternal pulsation
- 3) 1st aortic area
- 4) Pulmonary area
- 5) Left parasternal pulsation
- 6) Right parasternal pulsation

- ❖ **Parts of the hand used in palpation:**

☞ IF YOU PRESS, YOU MISS

☞ لو ضغطت ع النبض اوي مش هتحمسه صح اوي



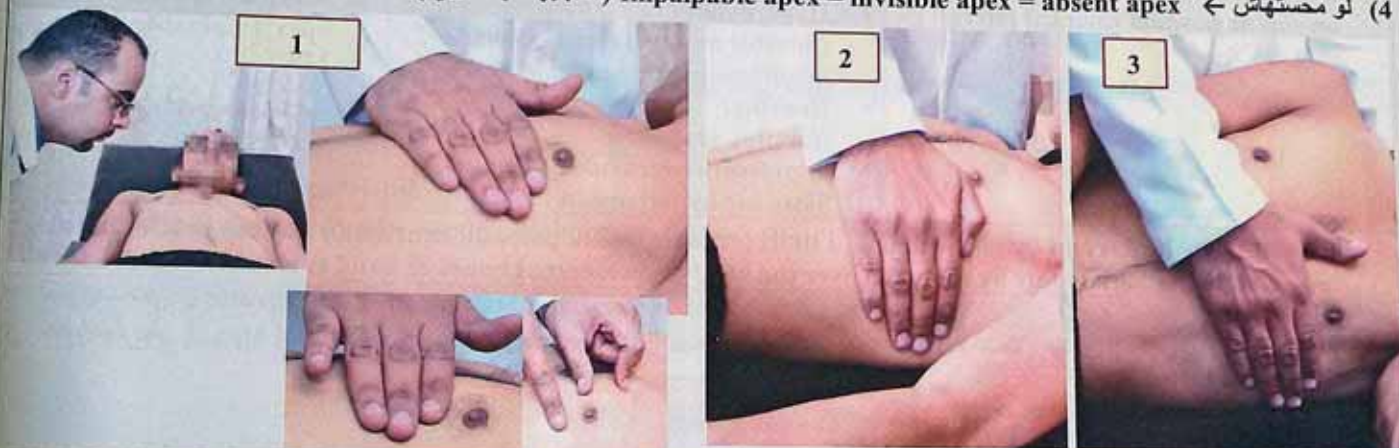
1. Apex :

❖ Comment on SACT.R

- ❖ **Definition:** outermost lowermost visible, strongest palpable pulsation point over the chest wall
- ❖ **Technique of examination:**

1. Site: patient in supine position, inspection to be confirmed by palpation

- (1) ابص tangentially على ال Puls. والعيان موقف نفسه , لو شافها وواضحة ←
 تحط عليها كافة ايدك اليمين finger pads (palmer surface of fingers) ← بعدين احدد اوضح Puls. بكذا صابع (finger tips) ←
 angle of Louis ← ال space اللي قصاها هو ال 2nd space ← (tip of index finger) ← اعد بالايه الثانيه بعد تحديد ال
- (2) لو Puls. مش شافها ومش واضحه ← حط كافة ايدك اليمين في ال posterior axillary line وارجع بيها لمكان الالاتومي بتاع ال apex في
 محاولة انك تحسها ← بعدين احدد اوضح Puls. بكذا صابع ← بصابع واحد ← اعد بالايه الثانيه
- (3) لو Puls. مش محسوسة في الوضع السابق ونته حاطط ايدك ← قول للعيان ينام على جنبه الشمال Left lateral ← لما تحس ال puls. ← حدد
 اوضح Puls. بكذا صابع ← بصابع واحد ← رجع العيان supine ← ورجع ايدك حوالي 2 cm ← اعد بالايه الثانيه
- (4) لو محسهاش ← Impalpable apex = invisible apex = absent apex (اسبابها مهمة اوي)



✓ Normally

- In adult : localized in left 5th space just inside MCL in supine position
- In children in left 4th space in MCL

⚠ Abnormally:

- LV enlargement: shifted outwards & downwards
- RV enlargement : shifted outwards

2. Area = extent = size: patient in supine position, inspection to be confirmed by palpation

- ♥ العيان نايم supine ← ابحث عن ال apex ← ثم تضع عليها صباعين لو غطوا ال apex تبقى localized , لو لسه تبقى diffuse
- ♥ لو ال Pulsation في space واحدة تبقى localized , لو أكثر من space تبقى diffuse



- Localized (One space, ≤ 3 cm i.e. well defined)

- LV enlargement : Localized (One space, ≤ 3 cm i.e. well defined)

- RV enlargement : diffuse (More than one space, > 3 cm i.e. ill defined)

3. Character: patient in supine position, by palpation only.

بص على ال apex ← حظ ايدك عليها

استخدم ال finger pads عشان تحس ال :

- Left lateral position NOT preferred to determine the character of the apex as it increases the apical pulsation and so misleading the original character.

Normal

Hyperdynamic

Heaving

slapping apex عشان تحس ال head of metacarpal bones ثم استخدم



Finger pads



Head of metacarpal bones

- ❖ Gentle non sustained tap formed by left ventricle, felt by the tips of finger on supine position then left lateral side

- ❖ LV enlargement : "forcible stronger than normal" :
 - **Hyperdynamic:** (قوية وبتجري) (forcible non sustained) in volume overload e.g. **MR, AR.**
 - **Heaving:** (قوية وبتطول) (forcible sustained) in pressure overload e.g. **AS, VSD, Systemic hypertension**

- ❖ RV enlargement:
 - Tapping (weak)
 - Slapping apex (weak apex + palpable S1) in MS

4. Timing with carotid by inspection & Thrill (organic lesion, palpable murmur): patient in left lateral position, by palpation only by the palm over the head of metacarpal bones & hand is moulded on chest wall

حظ ايدك على ال carotid عشان تعمل timing

حظ ايدك على ال mitral area بال head of metacarpal bones وخلي العيان ينم على جنبه الشمال عشان تزود ال Thrill



- No thrill
- Systolic thrill in MR (20%)
- Diastolic thrill in MS (80%)

5. Rhythm :

- Regular
- Irregular (AF or multiple Extrasystole)

6. Rate (apical) :

- Not usually done apically in normal
- Done in irregularity to determine pulsus deficit

7. Rocking : named after Rock & Roll dance

apex على ال و عنيك على ال carotid وشوف مع طلعة ال carotid يوجد bulge or retraction وحدد RV or LV



- Left or internal rocking (LVE):
 - Systolic bulge with left parasternal retraction; anti-clock wise rotation
- Right or external rocking (RVE):
 - Systolic retraction with left parasternal bulge: clock rotation

❖ **N.B.: Other abnormalities of the Site of the apex:**

1) Upward displacement:

- A. Supradiaphragmatic : collapse or fibrosis in upper lobe of Lt lung.
- B. Diaphragmatic : paralysis of Lt copula of diaphragm.
- C. Infradiaphragmatic : tense ascites, tumors & pregnancy

2) Downward displacement:

- Thin individuals with long chest , Emphysema "COPD" & Visceroptosis.

3) Inward "to the Rt." displacement:

- Cardiac causes : congenital dextrocardia
- Lung causes :
- Right sided fibrosis & collapse
- Left sided pleural effusion & pneumothorax

4) Outward "to the Lt." displacement:

- A. Cardiac causes : RV enlargement
- B. Lung causes:
 - Left sided fibrosis & collapse
 - Right sided pleural effusion & pneumothorax.

5) Outward & downward displacement:

- LV enlargement

6) Double apex :

- Ventricular aneurysm :systolic – diastolic pulsation (paradoxical)
- Hypertrophic cardiomyopathy : systolic – systolic pulsation

7) Impalpable apex "invisible apex" "absent apex":

A. Cardiac :

- Pericardial effusion, constrictive pericarditis
- Weak contraction (myocardial infarction)
- Dextrocardia

B. Chest causes :

- Obesity, behind a rib
- Emphysema, left sided pneumothorax or Pleural effusion

◆ Area	2. Suprasternal	3. 1 st Aortic Area	4. Pulmonary Area	5. Right Parasternal	6. Left Parasternal
◆ Site	▪ In suprasternal notch just above manubrium sterni	▪ Right 2 nd space within right parasternal line	▪ Left 2 nd space within left parasternal line	▪ 3 rd & 4 th right spaces within right PSL	▪ 3 rd & 4 th left spaces within left ParaSternal Line
◆ Inspection	▪ Look on suprasternal notch tangentially والعيان نايم أو قاعد	▪ Look tangentially while asking the patient to hold breath			
◆ Pulsation palpated by	▪ By tip of the index & middle finger perpendicular to suprasternal notch while the patient holding his breath in semi sitting position	▪ Placing the tips of the curved middle three fingers in the (aortic or pulmonary) area while the patient is holding breath	Puls. ال بعد كده تحس ال space وحد ال بال tips ♥		
◆ Causes of pulsation; Source	▪ Normal in thin people. ▪ Abnormally in same causes of big pulse volume	▪ Aortic dilatation or systemic hypertension	▪ Pulmonary artery dilatation or pulmonary hypertension	▪ Right atrium enlargement ▪ Huge Left atrium	▪ Right Ventricle enlargement ▪ Huge Left atrium
◆ Thrill by: by roots of fingers ◆ Timing against carotid pulse	❖ In neck "carotids": ▪ Propagated from heart base : AS accompanied by thrill over heart base ▪ Initiated in carotid itself "carotid shudder": AR, no thrill over heart base.	○ AS (systolic + propagated to neck), thrill increases in leaning forward position & expiration ♥ يميل للأمام ويخرج نفسه ويكتمه ○ Continuous : PDA	○ PS (systolic) ○ Continuous : PDA		○ VSD (systolic)
◆ Others	❖ Oliver's sign or vascular tracheal tug or tracheal tug with pulsation or true tracheal tug : • Descent of cricoid cartilage with ventricular contraction in cases of aortic aneurysm as aortic arch overrides the left main bronchus • Patient head is extended, the tip of index is placed below cricoid cartilage with slight pressure upwards → sudden jerky downwards movement of trachea (tugged) sufficiently to squeeze the finger can be felt with each heartbeat (with systole).	❖ Palpable Sound : diastolic shock = palpable A2 : • Aortic aneurysm e.g. in syphilitic AR • Systemic hypertension • Coarctation of aorta	❖ Palpable Sound : diastolic shock = palpable 2nd (P2) heart sound (↑S2): • In pulmonary dilatation due to pulmonary hypertension = grade 3 MS ♥ ممكن تحس في المنطقة ديه Palpable sound وده معناها ان ال ↑S2 ويطلق عليه diastolic shock لان مع ال diast. → Shock خبطة قوية		



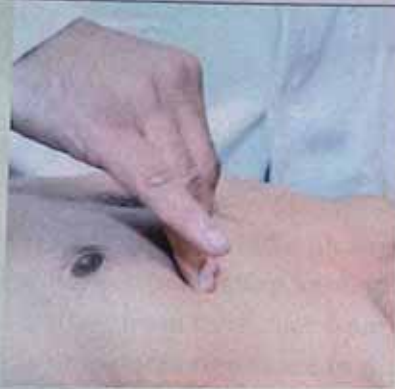
❖ Examination of areas :

✎ Inspection of pulsations :

♥ Epigastric → Apical → left parasternal → right parasternal → aortic → pulmonary → suprasternal pulsation

❖ لأن كل ال Puls. ترى tangentially لذلك انزل مرة واحدة وبص عليها بهذا الترتيب والعيان موقف نفسه :

✎ زني حرف S على العيان

❖ Pulsation		❖ Thrill & timing	
1. Suprasternal :			
			In neck "carotids"
2. 1 st aortic area			
			
3. Pulmonary area			
			
4. Left parasternal area:			
			
5. Right parasternal area:			
			

7. Epigastric Pulsation



❖ Site : below costal angle

❖ Inspected : look tangentially while asking the patient to hold breath
المريض يوقف نفسه

❖ Pulsation palpated by placing the extended hand in the epigastric area with fingers in the subcostal angle while the patient is holding his breath; try to feel the pulsation with the finger pads
ريح إيدك على البطن وزق صوابك في الـ epigastrium

❖ Source of pulsation:

○ From above: RV enlargement

○ From below : Aortic pulsation

○ From right side : Hepatic pulsation

❖ Confirmed by:



▪ Ask the patient to take deep breath and hold; pulsation get stronger

▪ Other signs of RVE

اجعل صوابك في مواجهة كتف العيان الشمال وقوله خذ نفس جامد واكتمه , هتلاقى تحت إيدك

▪ Hand is put just above or below the umbilicus slightly to the left of the midline to feel the aortic pulsation

▪ Increased with bimanual examination while the patient holding his breathing :

إيدك الشمال خلف العيان خلف آخر ribs
إيدك اليمين تحت الـ right costal margin واضغط

❖ Causes of pulsation; source:

▪ Causes of RVE:

1. Pulmonary hypertension
2. Pulmonary stenosis

1. The normal aortic pulsation is frequently visible in the epigastrum in thin people.
2. Same causes of prominent arterial pulsation especially AR

1. Normal thin people

2. TR:

○ The Most important cause of hepatic pulsation

3. TS

4. Pericardial diseases :

• Constrictive Pericarditis, Pericardial effusion

5. Liver disease e.g. hemangioma

❖ What are differences between TR & TS hepatic pulsations ?

❖ TR

○ Systolic hepatic pulsation (ventricular = arterial)

○ Coincide with S1

○ It's due to regurge of blood from right ventricle to IVC during systole

❖ TS

○ Presystolic hepatic pulsation (atrial = venous)

○ Coincide with S2

○ It's due to forcible atrial contraction just before systole

II. Percussion

❖ Types of percussion:

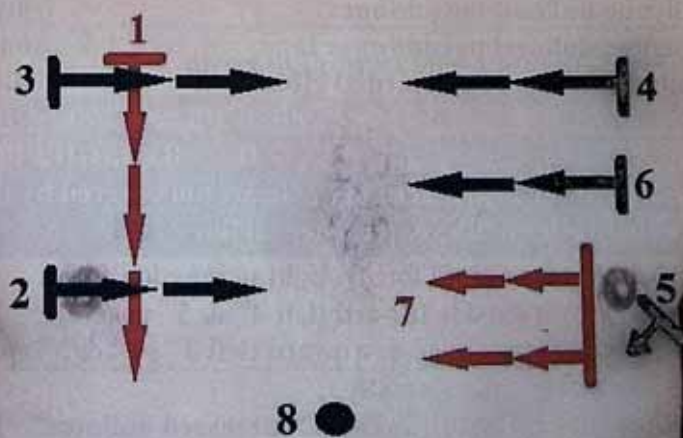
1. **Heart** : Heavy percussion (4-5 cm depth), except bare area by light percussion
2. **Lung** : Light percussion, except back by heavy percussion
3. **Abdomen** : Light percussion, except upper border of liver by heavy percussion

❖ Rules of percussion:

- A. The hand is firmly coapted to the surface
- B. Percussion is done on the middle phalanx or in interphalangeal joints (pleximeter) by the tip of the middle phalanx of the other percussing hand (plexor)
- C. Percussion is done from wrist, not from elbow
- D. Percussion is done from resonance to dullness, except Kronig's isthmus
- E. Percussion is done parallel to the border of dullness
- F. Don't damp the note

❖ Cardiac Percussion: Areas of Percussion

1. Upper border of the liver & Tidal Percussion
2. Right border of the heart
3. Aortic area (right 2nd space)
4. Pulmonary area (left 2nd space)
5. Outside the Apex
6. Waist of the heart (3rd left space)
7. Bare area (left 4th & 5th spaces, light percussion)
8. Lower end of sternum (light percussion)



I. BY HEAVY PERCUSSION:**1. Hepatic dullness (upper border of the liver): Tidal Percussion (TP)**

- ❖ Definition of TP: heavy percussion from the front in the RT MCL from above downward or back (Scapular line), after getting the 1st dull space, ask patient to take deep inspiration & re-percuss again:

1. If dullness changes to resonance: infradiaphragmatic = +ve TP e.g. liver

✓ Normally upper border of the liver in right 5th space MCL

⚠ Abnormally:

- Lower level of dullness (below right 5th space MCL): emphysema
- Higher level of dullness: causes of right lung base dullness (see discussion)

2. If dullness persists: supradiaphragmatic or diaphragmatic paralysis to differentiate percuss again in one space above, while the patient is holding his breath in deep inspiration:

A. If dullness changes to resonance: supradiaphragmatic dullness e.g. chest causes (pleural effusion, consolidation, collapse, fibrosis) (-ve TP)

B. If dullness persists: diaphragmatic paralysis due to upward movement of paralyzed diaphragm by negative intrathoracic pressure created by inspiration (reversed TP)

❖ So the value of TP:

- Differentiate between supradiaphragmatic & infradiaphragmatic dullness
- Indicate that diaphragm is freely mobile
- Reversed TP = diaphragmatic paralysis

2. Right border of the heart:

❖ Above the upper border of the liver by one space then move parallel to right border of the sternum, perpendicular to the rib, and percuss from outside right MCL medially.

✓ Normally: no dullness on right side of sternum

⚠ Abnormally: dullness: right atrial enlargement, pericardial effusion, dextrocardia, chest causes, elevated diaphragm (subphrenic abscess).

3. Upper border of the heart: Base of the heart:**A. 1st Aortic Area****B. Pulmonary Area**

❖ Detect the right 2nd space and then Percuss vertically from outside RT MCL towards the aortic area

❖ Detect the left 2nd space and then Percuss vertically from outside LT MCL towards the pulmonary area

❖ Percuss each space from lateral to medial from outside MCL to the sternum,

✓ Normally resonant, abnormally dullness occurs in:

1. Aortic dilatation (pulsation), aortic aneurysm
2. Post-stenotic dilatation (AS): no pulsation

1. Pulmonary artery dilatation (pulsation)
2. Post-stenotic dilatation (PS): no pulsation

3. Pleural effusion, chest causes, elevated diaphragm (subphrenic abscess)
4. Pericardial effusion: has a shifting dullness

❖ Shifting dullness:

- Definition: dullness on pulmonary area while patient is lying flat which disappears on sitting
- Cause: pericardial effusion
- Significance: differentiate between pericardial effusion & pulmonary dilatation as a cause of dullness in pulmonary area
- Technique: ask patient to sit down & re-percuss the dullness will change to resonant due reshaping of cardiac borders caused by effect of gravity on the fluid

4. Left border of the heart:**A. Apex:****B. Waist of heart: 3rd left space:**

❖ Detect the apex by palpation.

❖ Percussing outside of apex from lateral to the apex

❖ Detect the left 3rd space and then Percuss vertically from outside LT MCL towards the sternum

✓ Normally: no dullness outside apex

✓ Normally: 1-2 finger dullness

⚠ Abnormally: dullness outside apex in: ventricular aneurysm, pericardial effusion & chest causes

⚠ Abnormally: dullness more than 2 finger = obliteration of the waist
○ Left atrium ++, chest causes.

II. BY LIGHT PERCUSSION:

1. Bare area of the heart; area of the heart not covered by the lung tissue (cardiac notch) in the left 4th & 5th spaces between midline & parasternal line

❖ Percuss the left parasternal line by light percussion from:

A. Classic way: From outside inward (left 4th & 5th spaces)

B. Alternative way: from above downward (left 3rd, 4th & 5th spaces)

✓ Normally: dull "impaired note"

⚠ Abnormally:

1. Stony dullness "Increased dullness": RV enlargement, pericardial effusion & chest causes

2. Resonant: emphysema, left pneumothorax, dextrocardia

2. Lower third of sternum by light percussion by direct or indirect percussion:

✓ Normally: resonant

⚠ Abnormally: dullness occur in RV enlargement.

1. Hepatic dullness by tidal percussion



- ♥ ايدك على ال MCL
- ♥ انزل من ال 2nd space لحد ما تلاقي ال dullness
- ♥ قول للعيان ياخذ نفس واكتمه واخبط تاني هتلاقي
- ♥ انها اصبحت resonance لو كانت فعلا liver
- ♥ حدد مكان ال dullness وعد بابيك التاتية

2. Right border of the heart



- ♥ نفس اللي فوق كاملا +
- ♥ اطلع one space فوق ال upper border of
- ♥ the liver ثم لف صباعك 90 درجة علشان يكون موازي لل right border

A. By heavy percussion

3. Upper border of the heart:

- 1st aortic area: right 2nd space
- Pulmonary area: left 2nd space



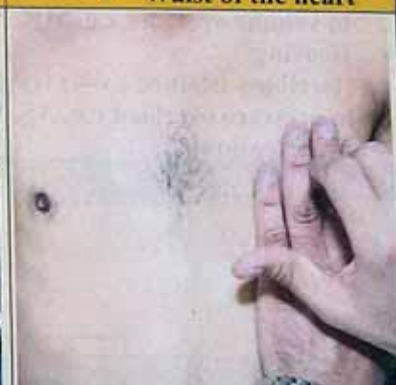
- حدد مكان ال space الاول وبعدين percuss من بره لجوه

4. Left border of the heart:

▪ Apex



▪ Waist of the heart



- ♥ خلي العيان يرفع ايده الشمال علشان يوسع
- ♥ تحدد ال apex بعينك ثم تبدأ من براها من
- ♥ عند ال AAL)anterior axillary line
- ♥ (وتدخل عليها في ال 5th space

- ♥ حدد مكان ال 3rd left space الاول
- ♥ وبعدين percuss من بره لجوه

By light percussion : lower third of sternum



- ♥ خبط على نهاية ال sternum خفيف direct or indirect

B. By light percussion: Bare are of the heart



- الطريقة ال classic من بره لجوه : حدد ال 4th & 5th space بعلامة ثم ضع يدك vertical وخط من الشمال (AAL) لليمين

- الطريقة ال alternative من فوق لتحت : حدد ال 3rd & 4th & 5th space واخبط من فوق لتحت

Remember

1. About percussion:

- A. Percussion of right border of the heart = tidal percussion + outside the right edge of sternum + aortic area
 B. Percussion of the base of the heart = upper border = aortic area + pulmonary area
 C. Percussion of the left border of the heart = outside apex + waist + pulmonary area

2. Signs of ventricular enlargement:

LV enlargement	RV enlargement
Causes	
- Volume overload: MR, AR - Pressure overload: AS	- Volume overload : TR, PR - Pressure overload : pulmonary hypertension
Apex	
- Shifted out & down - Localized (in 1 space) - Hyperdynamic: (قوية وبتجري) (forceful non sustained in volume overload e.g. MR, AR) - Heaving: (قوية وبتطول) (forceful sustained in pressure overload e.g. AS, VSD, Systemic hypertension)	- Shifted out - Diffuse (more than 1 space) - Tapping (weak) - Slapping apex (palpable S1) in MS
Others (bulge, 2 pulsation, 2 dullness)	
	- Precordial bulge - Epigastric pulsation - Left parasternal pulsation - Increase dullness "stony dullness" over bare area - Dullness over lower 1/3 of sternum
Rocking	
- Left or internal rocking: - Systolic bulge with left parasternal retraction; anti-clock wise rotation	- Right or external rocking: - Systolic retraction with left parasternal bulge; clock wise rotation

3. Signs of ventricular failure:

❖ Signs of RV failure; systemic congestion	❖ Signs of LV failure; pulmonary congestion
- Congested pulsating neck veins - LL edema - Enlarged tender pulsating liver - Ascites - Tricuspid gallop & functional tricuspid incompetence murmur: it's specific for right ventricular failure	- Apical gallop - Pulsus alternans - Fine bilateral basal Crepitations

4. Signs of atrial enlargement:

❖ Right atrium	❖ Left atrium
Causes	
TVD (TR غالباً + MVD)	MVD (stenosis or regurge)
Site	
Right parasternal area	3 rd left space (waist)
Signs	
Dullness in right parasternal area	Obliterated waist of the heart (dullness more than 2 finger)

5. Signs of arterial dilatation:

❖ Pulmonary artery	❖ Aorta
Causes	
Pulmonary hypertension (MS)	Aortic aneurysm & post-stenotic dilatation
Site	
Pulmonary area	1 st aortic area
Signs	
Pulsations, Diastolic shock & Dullness	Pulsations, Palpable S2, thrill & Dullness

III. Auscultation

1. Auscultation for :

- I. Heart sounds (S1 : over the Apex , S2 : over the Pulmonary area)
- II. Additional heart sounds (Systolic clicks; ejection click & opening snap)

III. Murmurs; Comment on the following: SACT.R

• Site of maximum intensity	• Area of propagation	• Character
• Timing	• Thrill & Grade	• Type (organic or functional)
• Relation to position , respiration & exercise		

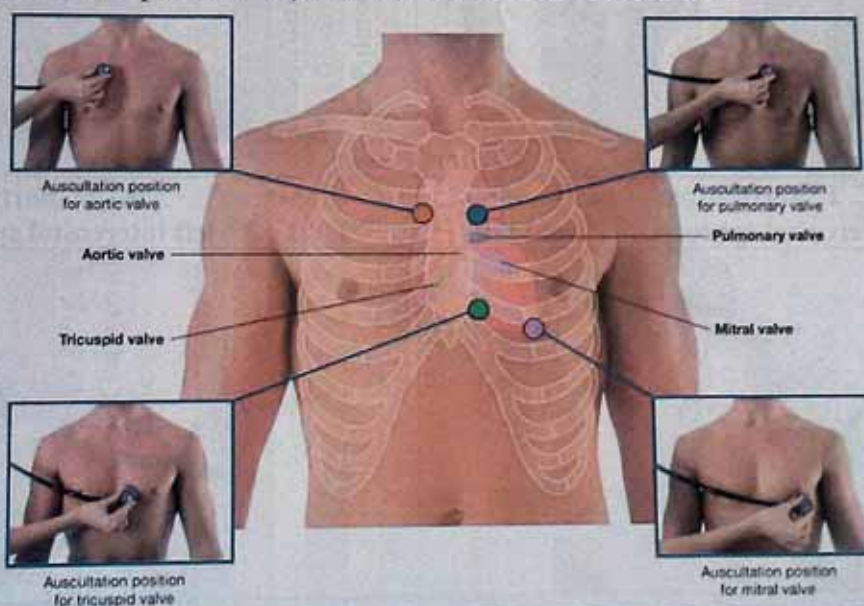
IV. Others heart sounds (metallic click)

2. Parts of stethoscope used for the sounds :

- Cone = bell (lightly applied): for low pitched sounds (murmur of MS, S3, S4).
- Diaphragm (firmly applied): for high pitched sounds : the rest of sounds.

3. The maneuver of auscultation: Z-Shaped auscultation: (M→T→P→A1)

- ❖ Technique : use the cone and auscultate the mitral area & tricuspid area then use the diaphragm and auscultate M→T→P→A1→A2→left parasternal→ axilla → root of the neck & over carotid arteries
- ❖ Site of auscultation:
- Pulmonary & tricuspid valve are anterior so, heard at their anatomical sites
- But aortic & mitral valve are posterior so, hear at the direction of blood flow



❖ قوانين ال Auscultation :

1. Time ← طول ما السماعة على العيان , ايدك على ال Carotid
 - Systole : مع ضربة (طلعة) ال Carotid
 - Diastole : مع نزلة (سحبة) ال Carotid
2. Site ← تحرك السماعة لحد لما توصل Site of maximum intensity
3. Character ← (cone → low pitch) (diagram → high pitch)
4. Position معين بيزود ال murmur اعمله
5. اوعى تقول تشخيص لو سالك الدكتور سامع ايه
6. اوعى تعلق على ال murmur تسميعاً من الكتاب

❖ Auscultation of:

1. Mitral area: (cone & diaphragm) = Apex : 5th left space just inside MCL

B. Left lateral position

A. Supine



2. Tricuspid area:
Lower end of sternum especially to the left.

3. Pulmonary area:
2nd left intercostal space, parasternal line



4. Aortic area

- A. 1st aortic area:
A1 : 2nd right intercostal space, parasternal line

- B. 2nd aortic area:
A2 : 3rd left intercostal space, parasternal line



5. Left parasternal area;
over 3rd & 4th intercostal space (for VSD)

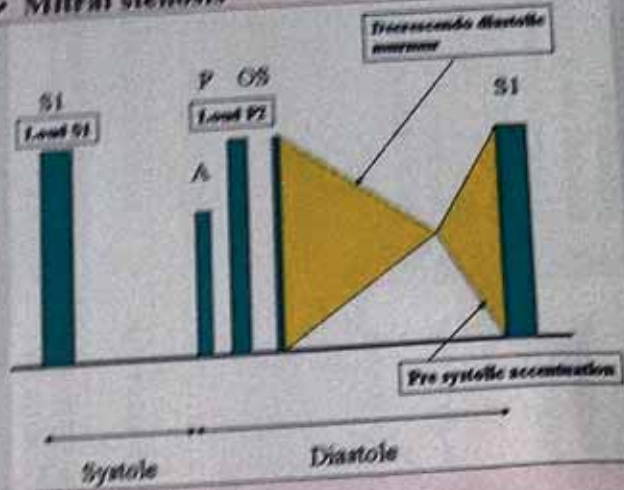
6. Axillary area :
propagation of MR murmur



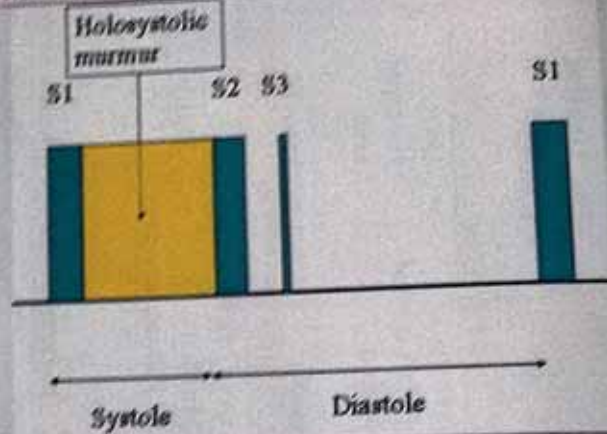
❖ Cardiac cases :

- Uni-valvular lesion: PS, AS, VSD, MT, TR, AR , MS
- Multi-valvular lesion : see later
- As a part of cardiac case: valve replacement case, Marfan syndrome & Noonan syndrome

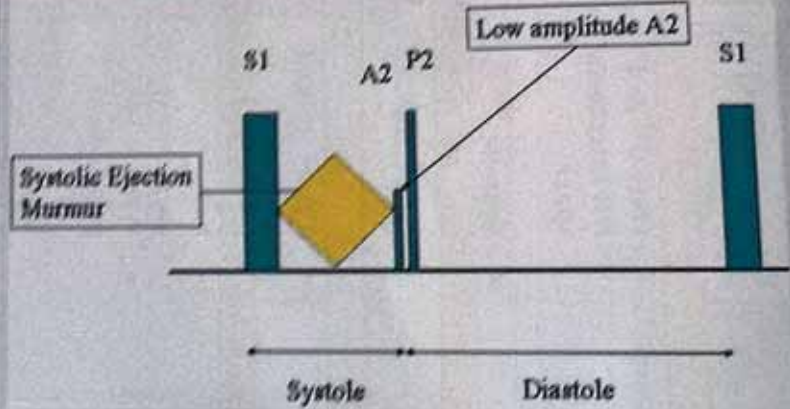
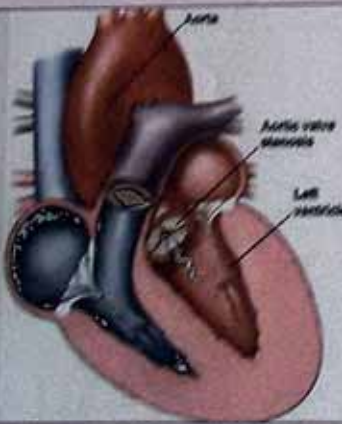
PS	AS	VSD	MR	TR	AR	MS
Apex Examination						
▪ Signs of RVE with heaving apex	▪ Signs of LVE with heaving apex	▪ Biventricular enlargement with hyperdynamic apex (HDA)	▪ Signs of LVE with HDA	▪ Signs of RVE with HAD ▪ General signs	▪ Signs of LVE with HAD ▪ General "Peripheral" signs	▪ Signs of RVE with slapping apex (brief, palpable S1) ▪ Diastolic shock (palpable S2 over pulmonary area); pulmonary hypertension
Thrill: Site & Time :						
▪ Over Pulmonary area	▪ Over A1 propagated to apex & neck (carotids) & ↑ on leaning forward	▪ Over 3 rd & 4 th left intercostal space	▪ Over apex ▪ ↑ in left lateral position	▪ Over tricuspid area	▪ No thrill over A1 ▪ Only in neck: AR	▪ Over apex ▪ ↑ in left lateral position
▪ Systolic	▪ Systolic	▪ Systolic	▪ Systolic	▪ Systolic	▪ Systolic	▪ Diastolic
Auscultation: Heart sounds.						
1. S1 over Mitral area						
	▪ Normal		▪ ↓ Muffled	▪ ↓ Muffled		▪ ↑ Accentuated
2. S2 over Pulmonary area						
▪ ↓ Muffled	▪ ↓ Muffled	▪ ↑ Accentuated (P.HTN)			▪ Normal or ↓ or ↑	▪ ↑ Accentuated, (P.HTN)
Auscultation: Murmur, Site of maximum intensity, Area of propagation, Character, Timing, Relation to position, respiration & exercise, Additional sounds						
▪ Pulmonary area	▪ 1 st aortic area	▪ Left parasternal	▪ Apex	▪ Tricuspid	▪ 2 nd aortic area	▪ Apex
▪ Tricuspid area (down) ▪ May be under left clavicle (up)	▪ Carotid (up) ▪ Apex (down)	▪ Whole precordium	▪ If anterior leaflet regurge to axilla ▪ If posterior leaflet : regurge to base & sternum	▪ Apex	▪ Apex	▪ Localized, not propagated
▪ Harsh ▪ Ejection systolic	▪ Harsh ▪ Ejection systolic	▪ Harsh ▪ Pansystolic	▪ Soft ▪ Pansystolic	▪ Soft ▪ Pansystolic	▪ Soft blowing ▪ Early diastolic	▪ Rumbling (cone) ▪ Mid diastolic presystolic with presystolic accentuation
▪ ↑ with inspiration	▪ Best heard with diaphragm, patient is: ○ Sitting ○ Leaning forward ○ Holding his breath in forced expiration (↑ with expiration) ▪ ↓ with exercise		▪ Patient : ○ On left lateral position ▪ ↑ with exercise	▪ ↑ with inspiration (Carvollo's sign)	▪ Best heard with diaphragm, patient position as AS ▪ ↑ with exercise	▪ Best heard with cone, patient position is: ○ On left lateral position ○ ↑ with exercise
• E.C. & S4	• E.C. & S4	• S3	• S3	• S3	• S3	• Opening snap



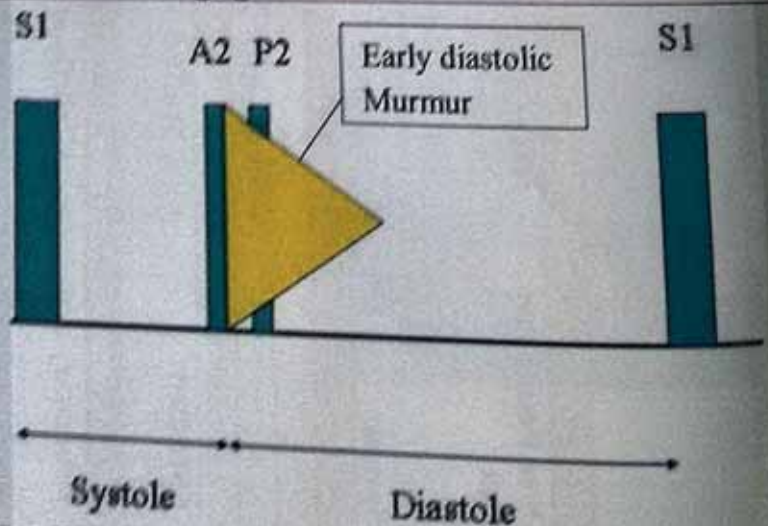
❖ Mitral regurg



❖ Aortic stenosis



❖ Aortic regurg



Special notes

1) LVE with MR, AS, AR

2) RVE with MS, PS, TR

3) BVE : VSD

- 4) All valvular lesion with systolic thrill in the specific area of valve **except** :
 • MS : Diastolic thrill • AR no thrill in A1, only in neck
- 5) All murmurs are systolic in time **except** AR (& PR), MS (& TS), are diastolic.
- 6) Any valvular **regurge** is soft in character & with **hyperdynamic** apex (volume overload)
- 7) Any valvular stenosis is harsh in character & with heaving apex (pressure overload) **except** MS is rumbling in character & with slapping apex
 ❖ N.B.: VSD : is harsh in character with hyperdynamic apex.
- 8) To detect S2 is accentuated (P.HTN) or not :
 ▪ Auscultate Over pulmonary area & compare between S2 over pulmonary area & S2 in aortic area:
 → S2 accentuated: when S2 gets higher in pulmonary area more than in aortic area

Marfan syndrome

❖ **Definition** : multisystem connective tissue disorder; autosomal dominant trait characterized by systemic affection :

1. Positive family history
2. Skeletal manifestations:
 - Long bone : disproportionate tall stature
 - Short bone : arachnodaetyly (long slender finger) proved by:
 - a. Thumb test
 - b. Wrist test
 - High arched palate
 - Flat foot; pes planus
 - Pectus excavatum or carinatum
 - Kyphoscoliosis
3. Eye: subluxation of lens , ectopic lentis
4. Chest: cystic diseases
5. Cardiovascular :
 - Aortic aneurysm
 - Dilated ascending aorta → **AR**
 - Mitral valve prolapse → **MR**



? How to diagnose Marfan ?

❖ Marfan syndrome:

- Positive family history ▪ Plus Skeletal manifestation ▪ Plus at least 2 Systems affected

? What other conditions may mimic Marfan syndrome = differential diagnosis ?

1. Marfanoid feature : skeletal manifestation only
2. Homocystinuria :
 - Patient have many symptoms and signs as tall stature & ectopic lentis
 - Aortic aneurysm is not a feature
 - Inheritance is recessive
3. Familial thoracic aortic aneurysm syndrome :
 - Has many feature of Marfan syndrome + autosomal dominant trait
4. MASS phenotype :
 - Has many feature of Marfan syndrome
 - But follow a more benign course

Valve replacement case

❖ Types of surgery:

A. Closed heart surgery	B. Open heart surgery
❖ Definition	Using heart lung machine
No heart lung machine	❖ e.g.
• Closed mitral commissurotomy (Valvulotomy)	• For stenosis : ○ Open valvulotomy, replacement by prothesis
	• For regurge : ○ Repair, replacement by prothesis
❖ Indications	• All valvular lesions except Isolated non calcific MS
• Isolated non calcific MS	❖ Scar
• Lateral thoracotomy (left inframammary)	• Median sternotomy
❖ Complications for both:	
1. Arrhythmias	2. Embolization
3. Valve regurge & restenosis	4. Postcardiotomy syndrome
5. Complications of artificial valves :	
• Infective endocarditis, Thromboembolism, Mechanical dysfunction & Hemolytic anemia	

❖ Types of prosthesis:

A. Tissue	B. Mechanical
○ Less durable : short life	○ More durable: long life
	❖ Others:
○ Less need for anticoagulant	○ Thrombosis : anticoagulant for life
○ Prosthetic valve endocarditis	○ Prosthetic valve endocarditis
	○ Hemolytic anemia

❖ How to know?

- **History** : of operation & which valve is replaced
- **Examination** : Scar of median sternotomy & Metallic sound may be heard

❖ How to deal?

→ As a cardiac case + answer the following questions:

1. Which valve is replaced ?

- ❖ Loud or metallic sound on the apex: (timing with carotid)
 - Metallic S1 → Mitral Valve Replacement
 - Metallic S2 → Aortic Valve Replacement

2. Complicated or not? لازم نسال العيان عن المضاعفات ولازم تذكرها بالاثبات او النفي

A. **Hemolytic anemia** : pallor + jaundice + fever

B. Thrombo-embolism :

- Symptoms of embolization e.g. hemiplegia
- Feel all arterial pulsation

C. Prosthetic valve endocarditis :

- Fever, clubbing, petechial hemorrhage & splenomegaly

3. The valve is functioning properly or not?

- If dysfunction : you will hear **murmur** of the lesion of the prosthetic valve “ stenosis or regurge”
- With the criteria of murmur of same rheumatic lesion of same valve

General "peripheral" signs of TR :

- Cyano-icteric face (mild jaundice from liver congestion & peripheral cyanosis from LOCP)
- Congested pulsating neck veins with systolic expansion
- Enlarged tender pulsating liver with systolic pulsation.
- Ascites precoc; ascites before Lower limb oedema.
- Percussion: right border dullness

General "peripheral" signs of AR :

A. Head & neck

1. Corrigan's sign : visible vigorous pulsation in carotid arteries in neck	2. De Musset sign : head nodding due to severe pulsation ❖ Other causes of head nodding: cerebellar ataxia, parkinsonism myoclonic epilepsy & tics	3. Systolic thrill "carotid shudder" : over carotids due to rapid blood flow
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B. Upper Limb :

- Wide pulse pressure : exaggerated difference between systolic & diastolic blood pressure (Normally 30 – 60 mmHg)
- Water Hammer pulse "Collapsing pulse"
- Capillary pulsation is demonstrated as follows :

▪ Quincke's sign : ○ Capillary pulsation in the nail bed : Pressing by finger on patient's nail just to cause blanching , the test is positive when the blanched area becomes alternatively red/blanch with each heart beat	▪ Transillumination by shining a pen torch through: Lobule of ear ▪ Forehead "Lighthouse sign" by scratch: blanching & flushing of forehead	▪ Fundus by ophthalmoscope : retinal capillaries are pulsating "Becker's sign" ▪ Lips by pressure by glass slide to produce blanched area ▪ Uvula "Muller's sign" by tongue depressor
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C. Lower Limb :

- Hill's sign : exaggerated difference between SBP in LLs & ULs more than 50 mmHg (normally, SBP in LLs is higher than ULs by about 10 – 20 mmHg)
- Pistol shot "Traub's sign": auscultation of a loud booming sound synchronous with each pulse beat over the arteries especially femoral due to sudden distension of collapsed artery
- Duroziez's sign : systolic & diastolic murmurs over the femoral artery if it is slightly compressed with the stethoscope.

❖ Significance of peripheral signs:

- Nonspecific present in hyperdynamic state
- Determine severity

❖ Absent peripheral signs in :

- Mild aortic regurge
- Associates stenotic valvular lesion:
- Double aortic lesion with predominant stenosis
- MS
- Heart failure
- Systemic hypertension

❖ **N.B.:** differential diagnosis between organic AS & function AS in AR

- Functional AS** systolic soft murmur localized over the 1st aortic area with normal S2 & no associated thrill
- Organic AS** (double aortic lesion): in which the murmur is harsh ejection systolic propagated to the neck and the apex, muffled S2 & associated with thrill and the patient complains of low COF symptoms.



☛ Pulmonary stenosis:

- May be due to malignant carcinoid tumour or due to congenital as Noonan syndrome
- ❖ **Noonan syndrome:**
 - Stunted growth, Subnormal mentality, congenital Skeletal deformity
 - Congenital heart disease (e.g. PS) & Special facial feature

☛ Effect of AF on mitral stenosis lesion:

1. Neck veins : absent a wave with systolic expansion
2. Pulse : marked irregularity
3. Pulsus deficit : more than 10 beats / min
4. Auscultation:

○ Loss of presystolic accentuation	○ Variable S1	○ Loss of S4 (no atrial contraction)
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5. Complications:
 - Thromboembolism (percentage of complication increase from 10% → 40 %)
 - Pulmonary oedema
6. ECG: absent P wave, marked irregularity

Multi-Valvular Affection:

Double mitral lesion = MR + MS:

- A. By history : palpitation + dyspnea
- B. By general examination : AF (irregular pulsation + systolic expansion of neck vein) + Orthopnea
- C. By local examination:
 - 1) Biventricular enlargement

❖ If regurge predominate	❖ If stenosis predominate
• Displaced apex	• Undisplaced apex

- 2) Signs of Pulmonary hypertension; signs :
 - General examination; neck vein: Giant a wave
 - Inspection : pulmonary pulsation, diastolic shock
 - Percussion: dullness in pulmonary area
 - Auscultation:

○ S2 ↑	○ Systolic ejection click	○ Systolic ejection murmur due to functional pulmonary stenosis (due to dilated pulmonary trunk)
○ Close splitting of S2		
○ Soft early diastolic murmur due to functional pulmonary regurge (Graham steel) (due to dilated pulmonary ring)		
○ S4 over the tricuspid area		

- 3) Murmur :

• Murmur of MR alone if murmur of MS is silent	• Murmur of both MR & MS
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- 4) Sounds:

▪ If regurge predominate	▪ If stenosis predominate
▪ ↓ S1	▪ ↑ S1
▪ S3 over the apex if regurge predominate	
▪ NO Opening snap	

Double aortic lesion = AR + AS

A. By history :

❖ If regurge predominate	❖ If stenosis predominate
• Palpitation • Chest pain • ± Pulmonary congestion	• LOCP Symptoms • Chest pain • ± Pulmonary congestion

B. By general examination :

❖ If regurge predominate	❖ If stenosis predominate
♥ Peripheral signs of AR especially : • Collapsing pulse, Wide pulse pressure , Corrigan's sign, Duroziez's sign	• Regular, slow rising pulse

C. By local examination:

❖ If regurge predominate	❖ If stenosis predominate
• LVE with hyperdynamic apex • ± Gallop rhythm (volume overload on LV)	• LVE with heaving apex
• S1 : normal • S2 : ↓ • Murmur of both: ○ AS: harsh ejection systolic murmur maximally over A1, propagating to neck ○ AR: soft early diastolic murmur, maximally over A2	

Aortic stenosis with Mitral stenosis

❖ By history :

AS	MS
LCOP	Dyspnea
• MS may be complicated by irregular palpitation + systemic congestion	

❖ By general examination :

- Regular pulse or irregular (as MS may be complicated by irregular palpitation; AF)
- Ankle edema (systemic congestion)

❖ By local examination:

- AS: harsh ejection systolic murmur maximally over A1, propagating to neck
- MS: rumbling mid diastolic murmur maximally over the apex, palpable S1

Mitral regurge with Aortic regurge:

❖ By history :

♥ Mitral regurge	♥ Aortic regurge
○ Palpitation	○ Palpitation
○ Dyspnea	○ Chest pain

❖ By general examination :

- Regular pulse
- Not uncommon to find elevated jugular venous pressure as indication of volume overload
- ± peripheral signs of AR

❖ By local examination:

- Signs of LV enlargement (displaced apex) & volume overloading (gallop rhythm)
- MR: soft pansystolic murmur maximally over apex & radiating to axilla
- AR: soft early diastolic murmur, maximally over A2

Mitral stenosis with Aortic regurge:

- By history → dyspnea, palpitation & chest pain
- MS masks signs of Peripheral signs of AR
- ↑ S1 & ↑ S2
- Murmur of AR
- Murmur of MS + Austin - flint murmur (relative murmur of MS in severe AR)

Mitral regurge with Aortic stenosis

- Murmur of MR
- Murmur of AS

Double Mitral with Double Aortic:

1. By history:

- Chest pain → AVD غالباً
- Pulmonary congestive symptoms (dyspnea) :
 - MVD غالباً
 - LVF صعب

- Palpitation → volume overload (MR, AR)
- LCOP → stenotic lesion (AS + MS)

2. Suggested findings:

- Peripheral signs of AR → AR
- Thrill on the base of the heart propagating to the neck → AS
- Apex hyperdynamic volume overload (MR, AR)
- Right ventricular pulsations → epigastric, left parasternal pulsation (associated MVD احتمال وجود)
- ↑↑ S1 + pansystolic murmur → double mitral lesion (silent MS)

3. N.B.:

- Silent MS = MS with no murmur due to:
 - A. High LV pressure: LVF.
 - B. Low LA pressure:
 - Severe pulmonary hypertension.
 - RVF.
 - C. In association with ASD.

❖ N.B.: ASD + Acquired MS = Lutembacher syndrome

Diagnosis Of Cardiac Case

I. Etiological diagnosis:	II. Anatomical diagnosis:	III. Functional diagnosis:
A. Rheumatic heart disease: ▪ Past history of rheumatic fever: ▪ Recurrent tonsillitis , pharyngitis , fever, arthritis ▪ Intake of anti-inflammatory (aspirin & steroid) ▪ IM injection of long acting penicillin ▪ Organic mitral stenosis, there is a thrill ▪ Double valve lesion (e.g. double mitral) ▪ Multivalvular = More than one valve affected (e.g. mitral & tricuspid)	I. Endocardial: valvular lesion e.g. AR, MS II. Myocardial (chamber enlarged): 1) RV enlargement : signs of RVE see before 2) LV enlargement: signs of LVE see before. 3) RA enlargement : dullness to the Rt. side of sternum 4) LA enlargement : obliteration of cardiac waist (better radiological diagnosis) 5) Pulmonary artery dilatation : ○ Pulsation & dullness in Pulmonary area ○ Diastolic shock & Accentuated S2 6) Aortic dilatation: ○ Pulsation and dullness in 1st Aortic area & Accentuated S2	❖ Compensation: I. Compensated: no symptoms or signs of failure II. Decompensated: in failure: 1. <u>Right Side Failure :</u> ○ Symptoms of systemic congestion ○ Congested neck veins ○ Enlarged tender liver ○ LL edema 2. <u>Left Side Failure :</u> ○ Symptoms of pulmonary congestion ○ Apical gallop ○ Pulsus alternans ○ Fine bilateral basal crepitations
B. Congenital: 1. Young age 2. Central cyanosis & clubbing 3. Associated congenital anomaly 4. Organic pulmonary lesion		❖ Complications: 1. Arrhythmia e.g. AF 2. Rheumatic activity & SBE 3. Pulmonary infection 4. Thromboembolism : hemiplegia
C. Others: ischemic, hypertensive, cardiomyopathy, systemic disease	III. Pericardial : pericarditis, pericardial effusion	
❖ E.g. a case of rheumatic heart disease, double mitral, tricuspid regurge with biventricular enlargement. The patient has right & left heart sided decompensation & complicated by atrial fibrillation (AF)		

Summary Of Cardiac Examination

General examination:

- Vital signs
- General overview
- Systemic overview
- Other systems except that of local exam

Local examination : cardiac examination

I. Combined inspection & palpation

- A. Precordial bulge
- B. Veins & scars
- C. Shape of the chest

D. Pulsations :

1. Apex pulsation : comment on SACT.R

- Site
- Area
- Character
- Thrill & Timing
- Rhythm
- Rate
- Rocking

2. Suprasternal pulsation

4. 1st aortic area

5. Pulmonary area

3. Epigastric pulsation

6. Right parasternal pulsation

7. Left parasternal pulsation

II. Percussion

A. By heavy percussion

1) Hepatic dullness by tidal percussion

3) Upper border of the heart:

2) Right border of the heart

• 1st aortic area

• Pulmonary area

4) Left border of the heart:

• Apex

• Waist of the heart

B. By light percussion

• Bare area of the heart

• Lower third of sternum

III. Auscultation

1. Heart sounds : S1, S2, S3, S4

2. Additional heart sounds : systolic clicks & opening snap

3. Murmur : comment on SACT.R

- Site of maximum intensity
- Area of propagation
- Character
- Timing
- Type : organic or functional
- Relation to position, expiration & exercise

4. Other heart sounds : (Pericardial rub, pericardial knock, crepitations & metallic click)

SCHEME OF CARDIOLOGY

❖ By history : أول شكوى

- ♥ Dyspnea → MS
- ♥ Irregular palpitation → AF with MVD (MS > MR)
- ♥ Low COP ± chest pain → AS
- ♥ Regular palpitation + chest pain → AR
- ♥ Systemic congestion → TR with MVD (MS > MR)

❖ By general examination:

- ♥ Blood pressure : ↑ systolic \ ↓ diastolic → volume > 60 mmHg → AR (signs على باقي ال)
- ♥ Pulse : irregular → AF with MVD (MS > MR)
- ♥ Orthopnea → MS
- ♥ Edema LL → TR with MVD (MS > MR)
- ♥ Tall stature → Marfan syndrome or marfanoid
- ♥ Stunted growth → congenital heart disease
- ♥ Cyanosis & clubbing → F4

❖ By Local examination:

I. Auscultation :

○ ابدأ السمع في ال 2nd aortic area وإسأل نفسك في murmur ولا لا :

B. لو انتة سامع Murmur , اعمله timing مع ال carotid	A. لو مش سميع murmur : ← روح على ال apex أكيد متلافي murmur : اعمله timing مع ال carotid
1. If diastole : AR	1) If diastolic → MS (localized)
2. If systole : → تحرك بالسماعة	2) If systolic → MR (anterior leaflet) → axilla
• ↑ towards carotid & A1 → AS • ↑ towards apex → MR • ↑ towards pulmonary area → PS • ↑ towards tricuspid → TR	

II. Inspection & palpation: مشيها مع التشخيص

III. Percussion: مشيها مع التشخيص

INVESTIGATIONS OF A CARDIAC CASE

I. Non-Imaging:

1. ECG:

• Types of ECG:

- Resting ECG **العيان نائم**
- Exercise ECG **بعجلة**
- Ambulatory ECG **يتحرك بيه**

• Value :

- To detect arrhythmia e.g. AF or extrasystole
- Chamber enlargement (hypertrophy) rather dilatation due to ischemia
- ⚠ N.B.: you have to know ECG changes of the following diseases:
 - Chamber hypertrophy, angina, myocardial enlargement & pericarditis

→ For more details about imaging, ECG, Clinical pathology, see Paraclinical Made easy for the author.



2. **Cardiac enzymes:** CPK, LDH, CPK-MB, Troponins; they increase in myocardial infarction
3. **Blood culture :** for infective endocarditis
4. **Blood tests** for identification of risk factors e.g. lipid profile for dyslipidemia
5. **Acute phase reactants** for general evidence of tissue damage in MI : leukocytosis, ↑ESR, ↑CRP

II. Imaging:

1) Chest X-ray:

- A. Plain X-ray : to detect chamber enlargement & pulmonary congestion,, pleural effusion valve calcification, prosthetic valve
- B. Contrast X-ray: barium swallow in lateral view in MVD to detect left atrial enlargement : indentation or posterior displacement of esophagus

→ For more details, see Paraclinical Made easy for the author.

2) Ultrasonography (US): أشعة تليفزيونية (موجات فوق صوتية)

❖ Technique:

- A. Intravascular US: جهاز الموجات فوق الصوتية لأوعية الدم
- B. Echocardiography → anatomy فحص القلب بالموجات فوق الصوتية (أشعة الإيكو) أو تخطيط صدى القلب

♥ Types of echocardiography:

○ Transthoracic echocardiography	○ Transesophageal echocardiography	○ Stress echocardiography
○ Contrast echocardiography	○ Doppler echocardiography	
○ Dobutamine induced echocardiography (pharmacological stress)		

♥ Value of Transesophageal echocardiography (TEE): indicated in all cardiac cases

▪ Thrombi : AF	▪ Vegetations : SBE	▪ Aortic aneurysm	▪ Valve replacement especially mitral & aortic
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C. Doppler US → blood flow & pressure فحص القلب بالدوبلر

D. Echo Doppler فحص القلب بالإيكو دوبلر

• Value : to detect

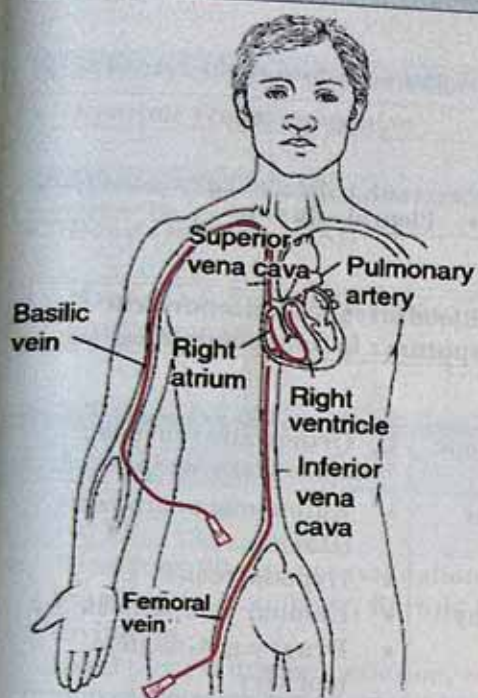
○ Valvular affection; which valve, what lesion & { severity of the lesion = indications of surgery}		
○ Chamber enlargement	○ Congenital abnormalities	○ Intracavitary masses; thrombus & tumour
○ Vegetation of endocarditis	○ Myocardial function by ejection fraction	○ Pericardial disease; effusion & constrictive pericarditis

3) Pulmonary angiography for pulmonary embolism

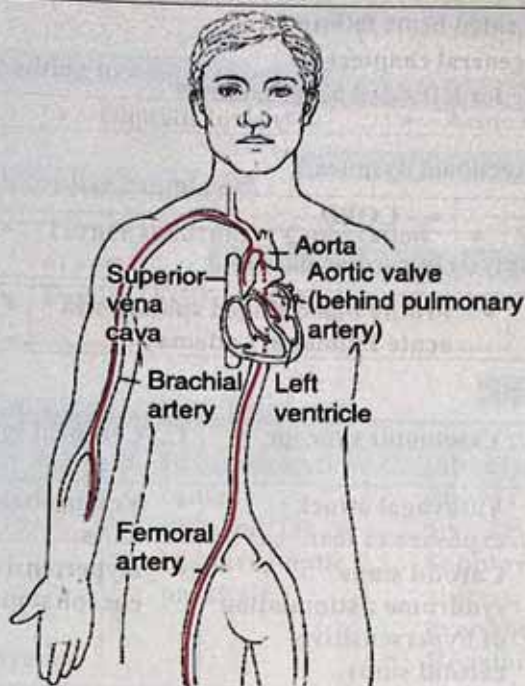
4) CT & MRI: in dissecting aorta

5) Cardiac catheterization = coronary angiography:

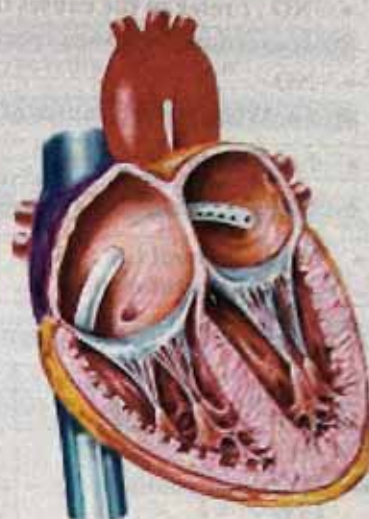
a. Right sided catheter:



b. Left sided catheter:



c) Trans-spetal technique



❖ Technique : Wire : long, malleable, very thin, hollow , radio opaque

❖ Approach :

A. Right sided catheter:

- Any vein → RA → Tricuspid valve → RV → pulmonary valve → pulmonary artery

B. Left sided catheter:

- Artery → aorta → aortic valve → left ventricle only

C. Mitral valve : trans-spetal technique : to reach LA:

- Right sided catheter : right atrium → pierce interatrial septum (artificial ASD) → left atrium → mitral valve

○ Indications:

A. Measure the pressure : pulmonary hypertension, pressure gradient

B. Take a blood sample : for oxygen saturation to diagnose shunt

C. Injection of contrast : cin-angiocardiology

○ Value:

- As US
- Coronary angiography.

6) Cardiac scan = Isotopic studies= Radionuclide imaging (radioactive Thallium 201):

Thallium blood flow

Stress

Rest



Exercise-induced
ischemic area

Fill-in 3 h later
shows viable
myocardium

○ Diagnosis of myocardial ischemia & infarction

○ Thallium is taken by healthy myocardium & not by the ischemic myocardium

○ Ischemic area appears as a filling defect (cold spot) at exercise & disappears at rest

○ infarcted area appears as a filling defect (cold spot) at rest

CLINICAL DISCUSSION

- ❖ Is orthopnea specific for left sided heart failure ?
- NO , (refer to the causes in general chapter)
- ❖ Is exertional dyspnea specific for left sided heart failure ?
- NO
- ❖ So, What are the causes of exertional dyspnea ?

• Left side heart failure	• COPD	• Pleural effusion
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- ❖ What are the causes of hemoptysis in cardiac patient ?

• Frank hemoptysis : MS , pulmonary infarction	• Frothy blood tinged sputum : in acute Pulmonary edema	• Blood streaked mucopurulent sputum : in pulmonary infection
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- ❖ What are the causes of syncope?

A. Cardiac syncope : due to low CO	B. Vasomotor syncope	C. Cerebral syncope	D. Orthostatic syncope : "postural syncope"
• AS • Acute heart failure e.g. AMI • Arrhythmias (tachy or brady) • Adams stokes attack	• Vasovagal attack : exposure to fear • Carotid sinus syndrome : stimulation of hypersensitive carotid sinus	• Verobasilar TIAs • Hypertensive encephalopathy	• Autonomic neuropathy (DM), • Hyponatremia • Lumbar sympathectomy • Drugs e.g. Ganglion blockers

- ❖ What are the cardiac causes of chest pain ?

▪ Myocardium : angina, Myocardial infarction	▪ Pericardium: Pericarditis, Pericardial effusion	▪ Endocardium : mitral valve prolapse
▪ Pulmonary infarction	▪ Dissecting aneurysm of aorta	▪ Pain of cardiac neurosis

- ❖ What are the causes of angina pain in young age ?

• Aortic valve disease	• Mitral valve Prolapse	• Prinzmetal angina
• Vasculitis	• Familial hypercholesterolemia	

- ❖ What are the causes of pressure symptoms of cardiac patient ?

- Left Atrium enlargement, pericardial effusion, aortic aneurysm

- ❖ What do you know about Ortner syndrome ?

- Hoarseness of voice due to Recurrent laryngeal nerve palsy from enlarged left atrium due to mitral stenosis

- ❖ What is the origin of emboli that patient may get & their effects?

❖ Symptoms of systemic embolization:

❖ Origin :	❖ Effects :
1) Atrium: Left atrium in MS especially when AF develops	1. Middle cerebral artery : embolic hemiplegia
2) Valve: mitral & aortic valves in SBE, prosthetic valves in valve replacement	2. Central retinal artery ; sudden blindness
3) Ventricle: Left Ventricle mural thrombus in Myocardial infarction	3. Coronary artery : chest pain (myocardial infarction)
4) Vessel: aortic atherosclerotic plaque in atherosclerosis	4. Superior mesenteric artery : abdominal pain (intestinal obstruction)
	5. Splenic artery : pain in Lt hypochondrium
	6. Renal artery : painless hematuria
	7. Peripheral artery : pulseless, pallor, pain, paresthesia & paralysis

- ❖ What are the palpable sounds you can feel?

• Palpable S1 in slapping apex in MS	• Palpable 2 nd (A2) heart sound in syphilitic AR, hypertension & COA	• Palpable 2 nd (P2) heart sound in pulmonary hypertension = grade 3 MS	• Palpable 3 rd or 4 th heart sound gallop
• Palpable rub in pericarditis			

❖ What are the causes of gynecomastia in cardiac patient ?

- Chronic hepatic congestion
- Drugs (Spironolactone, digitalis, cimetidine)

❖ What are the causes of nausea & vomiting in a cardiac patient ?

- | | | |
|------------------------------|----------------------|-------------------------------|
| • Systemic venous congestion | • Digitalis toxicity | • Acute myocardial infarction |
|------------------------------|----------------------|-------------------------------|

❖ What are the inherited diseases with cardiac complaint?

• Down syndrome : VSD	• Turner syndrome : Coarctation of aorta	• Marfan syndrome : aortic incompetence
• Myotonia & Friedreich ataxia : cardiomyopathy	• Familial hypercholesterolemia	

❖ What are the differential diagnosis of right lung base dullness ?

A. Supradiaphragmatic causes : -ve TP	B. Diaphragmatic causes : reversed TP	C. Infradiaphragmatic causes : + ve TP	D. Chest wall causes :
○ Basal lung disease : consolidation, bronchiectasis, collapse, fibrosis & cavitation ○ Basal pleural disease : effusion, empyema, hydropneumothorax & pleural Fibrosis ○ Cardiac causes : dextrocardia, huge pericardial effusion & huge cardiomegaly	○ Diaphragmatic paralysis	○ Subphrenic abscess, amebic liver abscess ○ Hepatomegaly, abdominal tumors ○ Marked ascites, pregnancy	○ Tumors of skin, subcutaneous tissue, muscles & ribs

❖ What are the Characters of cardiac edema?

❖ Occurs in the dependent parts of the body : ▪ Ankle oedema : in ambulant patient ▪ Sacral oedema : in bed ridden patients	❖ Bilateral : but one side may be affected more (<i>unequal edema</i>) due to : ▪ Deep venous thrombosis (DVT) ▪ Postural, in patients sleeping on one side
❖ Pitting	❖ Associated with other features of cardiac diseases
❖ Oedema of lower limbs always precedes appearance of ascites except in two conditions in which ascites occurs first Ascites precox (ascites before edema) occur in : ▪ Tricuspid valve disease : TR - TS ▪ Pericardial disease : pericardial effusion & constrictive pericarditis	

❖ What is the difference between rheumatic fever, rheumatic heart & rheumatic activity?

- Rheumatic fever : is the first attack of rheumatic fever
- Rheumatic heart : it is the scar left by the rheumatic in the form of valvular affection
- Rheumatic activity: it is rheumatic fever on top of rheumatic heart or the attack of rheumatic other than the 1st attack.

❖ What is the mechanism of PND; paroxysmal nocturnal dyspnea?

• It's pathognomonic to Left side failure:

• Mechanism :

- A. **During sleep** → ↑ parasympathetic and ↓ sympathetic tone leading to :
- Inhibition of respiratory center → hypoxia with hypercapnia till a critical level at which there is sudden stimulation of respiration.
 - Bradycardia, decreased contractility leading to decreased cardiac output leading to exaggeration of heart failure.
- B. **It occurs after 1-2 hours**, the time needed for redistribution of oedema fluid from lower limbs to the lungs

❖ What is the pathogenesis of cardiac dyspnea ?

A. Mechanical factors :

1. ↓ alveolar compliance secondary to interstitial oedema
2. ↓ lung compliance secondary to concomitant pleural effusion & elevated diaphragm (ascites, enlarged liver)
3. Oedema of bronchial mucosa

B. Nervous factors :

1. Activation of Herring – Breuer reflex:	2. Churchill- cope reflex:
• It is a normal slowly adapting reflex where impulses arise from stretch receptors, in the terminal air passages at the end of inspiration leading to reflex inhibition of inspiration & passive relaxation, i.e. expiration • In left side failure, interstitial oedema activates this reflex leading to rapid shallow respiration	• It abnormally rapid adapting reflex which occurs only in pulmonary congestion with congested pulmonary vessels that stimulate J-receptors (Juxta-alveolar receptors) leading to reflex stimulation of respiratory center in brain stem

C. Chemical factors:

- Hypoxia, hypercapnia, acidosis are stimulant to respiration.
1. Hypoxia secondary to pulmonary congestion
 2. Hypercapnia : occurs in severe acute pulmonary congestion i.e. acute pulmonary oedema
 3. Acidosis: secondary to tissue hypoxia leading to lactic acidosis

FREQUENTLY ASKED QUESTIONS FOR MID & END ROUND EXAM

Test yourself!

1. What are the cardiac causes of jaundice?	2. What are the cardiac causes of fever?
3. What are the cardiac causes of clubbing?	4. Define apex the heart?
5. What are causes of out displacement of cardiac apex?	6. What shifts the apex of the heart "out & down"?
7. Systolic bulge of the apex is caused by?	8. Systolic retraction of the apex is caused by what?
9. What are the differences between localized & diffuse apex?	10. What are the characters of the apex?
11. What are causes of thrill over the apex?	12. What are the causes of left parasternal pulsation?
13. What is the cause of pulmonary pulsation?	14. What is meant by diastolic shock?
15. What are the causes produce thrill on cardiac base?	16. What are the causes of epigastric pulsations?
17. What are the causes of aortic pulsation?	18. What is the most important cause of hepatic pulsation ?
19. What is the difference between systolic & presystolic hepatic pulsation?	20. What are the causes of invisible – impalpable apex?
21. Causes of dullness outside right sternal border (right border of the heart)?	22. How to differentiate between pericardial effusion & pulmonary dilatation as a cause of dullness in pulmonary area
23. What are causes of dullness outside apex?	24. What are the causes of enlarged bare area with stony dullness?
25. What is the extent of bare area?	26. What is meant by obliterated waist?
27. What is meant by tidal percussion?	28. What is meant by reversed tidal percussion?
29. What are causes of accentuated 1 st heart sound?	30. What are causes of variable S1?
31. What are the causes of pansystolic murmur?	32. What are causes of ejection systolic murmur?
33. What are the causes of diastolic murmur?	34. What is the Carvallo's sign?
35. What are the grades murmur?	36. What are the peripheral signs of TR?
37. What are the peripheral signs of aortic regurge?	38. Investigation of cardiac case?
39. What are signs of right ventricular enlargement?	40. What are signs of left ventricular enlargement?
41. What are the causes of volume overload on LV?	42. What are the causes of pressure overload on LV?
43. What are the Causes of right ventricular enlargement?	44. What are the causes of pulmonary hypertension?
45. What are the signs of pulmonary hypertension?	46. What is the definition of rheumatic fever?
47. What are criteria of rheumatic fever?	48. How to diagnose rheumatic fever?
49. What is the difference between rheumatic fever, rheumatic heart & rheumatic activity?	50. What is the treatment of rheumatic fever?
51. What are the complications of rheumatic heart disease (MS, AS, MR)?	52. What is the treatment of rheumatic heart disease?
53. All questions answered in the clinical discussion are very important.	

O.S.C.E QUESTIONS

▪ Inspect & palpate heart	▪ Inspect & palpate pericordium	▪ Inspect & palpate the apex
▪ Percuss & auscultate the heart	▪ Comment on the base of the heart	▪ Comment on the apex
▪ Search for signs of ventricular enlargement	▪ Peripheral signs of AR	▪ Auscultate heart (AS, AR, MS, MR, DM, metallic sound)

Mitral stenosis

❖ Personal history:

❖ Complaint :

- Shortness of breath of one week duration.

❖ History of present illness:

- The condition started 15 years duration by gradual onset and progressive course of **dyspnea**. The patient experienced **exertional dyspnea** on less than ordinary effort, on climbing the 1st floor not associated with orthopnea or PND. The patient sought medical advice, investigated by chest X-ray, ECG and ECHO, diagnosed as tight mitral stenosis and treated by mitral valvotomy.
- The patient remained symptom-free for 1 year after operation then he developed **exertional dyspnea** again on ordinary effort not associated with orthopnea or PND. This dyspnea was associated with gradual onset of **irregular palpitation** increased with exertion.
- The patient sought medical advice, investigated by chest X-ray, ECG and ECHO, and treated digitalis and lanoxin.
- The patient was quite well till one week ago when he re-developed **exertional dyspnea** again on ordinary effort associated with orthopnea or PND.
- No symptoms of **low COP**. No symptoms of **systemic congestion**.

❖ Past history:

- There is past history of **rheumatic fever** Since he was 14 years old, manifested by fever and arthritis, investigated by CBC, ESR and ECHO, treated by aspirin and the condition relieved and he was advised to take long acting penicillin for life and it was recurrent several times.
- No DM no HPN. No past history of operations or drugs.

❖ Family history

- No consanguinity, No common diseases in the family, No similar condition in the family.

❖ General exam

- **Temperature:** 37°
- **Blood pressure:** 110/70.
- **Pulse:** markedly irregular pulse, 75/minute, variable volume, pulsus deficit >10, vessel wall is not felt, equal on both sides with intact peripheral pulsation radio-femoral delay with .
- Average **built**. No **cyanosis**, **pallor** or **jaundice**.
- No special **decubitus** (the patient is lying free flat comfortable in bed).
- No **oedema L.L** , No **clubbing**. **Neck veins** are pulsating not congested.

❖ Local exam

1. Inspection and palpation

- Left inframammary thoracotomy scar of mitral valvotomy. Normal shape of chest.
- No dilated veins on chest wall. No pericardial bulge.
- Regarding pulsations :
- **Apex:**
 - Markedly irregular apex, 90/min, lies in left 5th space MCL, localized, slappy in character, with no thrill or rocking movement.
- Otherwise, apart from weak epigastric pulsations originating from aorta, no other visible or palpable pulsations.

2. By palpation only :

- **Pulsations** the same as above.
- **Palpable sound:** there is palpable 1st heart sound (slappy apex) but no diastolic shock.
- **No thrill** either on apex, left parasternal area or on base.

3. Percussion :

- **Hepatic dullness** in the Right 5th space MCL, no dullness in the right parasternal area, both aortic and pulmonary areas are resonant, preserved waist of the heart, no dullness outside the apex, lower end of sternum is impaired note with dull bare area.

4. Auscultation

- **1st sound** accentuated.
- Diastolic rumbling **murmur**, localized over the apex, increased on lying in the left lateral position, it's grade III/VI with no thrill.
- **Opening Snap** may be heard!!

■ N.B :

- In A.F.: Notice the following difference: Variable 1st heart sound with no presystolic accentuation of the murmur.

○ Discussion :

- ? What is your diagnosis ?
- Rheumatic heart disease, in the form of isolated mitral restenosis, the patient is compensated and complicated by AF.
- ? Why Rheumatic? → from history.
- ? Why MS? → from history, exam (irregular pulse, scar, slappy apex, auscultation).
- ? Why Compensated? → from (absence of symptoms and signs of systemic congestion).
- ? Why Complicated by AF? → (irregular pulse with marked irregularity and pulsus deficit >10).
- ? Why is S1 accentuated in the patient with MS?
 - ☐ Fibrosis of the mitral cusps.
 - ☐ Forcible closure of the mitral cusps : They are displaced downwards due to high LA pressure & The mitral valve is opened as wide as possible during the diastole.
- ? In which condition is S1 diminished over the apex in MS ?
- ☐ In cases of MS which are associated with MR (double mitral), or in cases of calcified mitral valve
- ? What additional sound can you hear in MS ?
- ☐ Mitral opening snap: وتشرحها للدكتور بالتفصيل
- ? How can you prove calcification of the mitral valve by one single investigation ?
- ☐ Chest X ray.
- ? When can you find MS with no murmur ?
- ☐ Silent Mitral Stenosis (MS with no murmur) due to:
 - A. High LV pressure: LVF.
 - B. Low LA pressure:
 - Severe pulmonary hypertension.
 - RVF.
 - C. In association with ASD.
- ? Can you hear also S3 over the mitral area in this patient ?
- ☐ No, because there is no LVF in pure MS, & there is no volume overload over the LV in pure MS
- ? So, when can you hear S3 over the mitral area in MS ?
- ☐ In cases of MS which are associated with MR (double mitral)
- ☐ In cases associated with aortic valve disease: AR & AS
- ? If the patient develops AF as a complication, what will you find clinically?
- ☐ See before.... (effect of AF on MS)
- ? What other complications of MS do you know ?
- ☐ Revise the complications from your theoretical book
- ? What other causes of MS do you know ?
- ☐ Revise the causes from your theoretical book
- ? Why is infective endocarditis not common in MS ?
- ☐ Due to weakness of the blood jet passing from the LA to the LV (infective endocarditis needs a high pressure gradient to occur).
- ? How could you differentiate between organic MS & Austin flint (relative MS) in AR?
- ❖ Organic MS: mid diastolic murmur with presystolic accentuation , ↑ S1, opening snap, diastolic thrill
- ❖ Austin flint in AR: mid diastolic murmur on apex with normal S1, no opening snap, no thrill.
- ? Why is PND (and acute pulmonary oedema) not common in MS ?
- ☐ Because MS is a gradual process allowing time for protective mechanisms to take place to ↓ further pulmonary congestion:
 - A. Reflex vasoconstriction of pulmonary arteries.
 - B. Anastomosis between pulmonary & bronchial veins
 - C. Development of interstitial pulmonary barrier.
 - D. Thickening of the walls of pulmonary vessels.

- ? What is meant by tight MS & what is its importance ?
- ☐ Tight MS is an indication for surgery & is diagnosed by:
- ☐ According to the stage: Stage II with dyspnea more than grade II, or Stage III, or Stage IV
- ☐ According to the echocardiography: Valve area less than 1 cm^2
- ? How would you proceed to manage this case?

A. Investigations

- ? What do you expect to find in the ECG of a patient with MS ?

1. ECG :
 - LA enlargement (P mitrale : m shaped P wave)
 - Pulmonary hypertension (P pulmonale : peaked P wave)
 - Arrhythmia (AF)

- ? What do you expect to find in the chest x-ray of a patient with MS ?

2. CXR for chamber enlargement.
 - LA enlargement (lateral view with barium), Pulmonary congestion, Dilated pulmonary artery, RVE. & Calcification of mitral valve.

- ? What is the most important investigation of MS ?

3. Echocardiography (investigation of choice, the most diagnostic, the most important) & Echo Doppler: anatomical, functional, aetiological, diagnosis and for detection of complication.

- ? How to calculate mitral stenosis index ?

4. Catheterization & angiography:

- Chamber enlargement.
- Mitral stenosis index = $\text{COP/LAP} \times 100 = 5/5 \times 100 = 100\%$ (< 25 % is tight MS)

B. Treatment

- a) Medical treatment

- Prophylaxis against IE & rheumatic activity.
- Treatment of complications e.g. HF, AF, infections ...

- b) Interventional (balloon mitral valvoplasty).

- c) Surgical (valvotomy or valve replacement).

- ? What would you add to your treatment if the patient with MS presented also with AF ?

- ☐ In addition to treating AF by anti - arrhythmic drugs, I would like to add anticoagulants for life

- ? What kind of surgery would you do for this patient with stenosed mitral valve ?

- ☐ Types of operations:

- Mitral commissurotomy: closed or open.
- Valve replacement: By a prosthesis (tissue or synthetic).

- ? What are the indications of valve replacement of that patient?

- ❖ Calcification

- ❖ Associated MR

- ❖ Tight MS (MS Index < 25 % , surface area < 1 cm^2 or severe manifestations)

- ❖ Recurrent stenosis after balloon dilatation or valvotomy .

- ? What are the surface anatomy of mitral valve?

- Site : between the LA & LV .
- Surface area : 4 - 5 cm^2 , if < $1 \text{ cm}^2 \rightarrow$ tight MS.
- Shape of mitral orifice : rounded or oval during diastole & slit like during systole.
- Axis : downward , forward & to the left.
- Components:
 - Fibrous ring.
 - 2 cusps (anteromedial & posterolateral)
 - 2 papillary muscles : arise from the ventricle, to control the cusps movement.
 - Chordae tendinae : arise from papillary muscles to both cusps.

Aortic stenosis

❖ **Personal history:.....**

❖ **Complaint:**

- Chest pain of ten days duration.

❖ **History of present illness:**

- The condition started ten days ago by retro-sternal constricting chest pain radiated to the left shoulder, increased by exercise, relieved by rest and lasted for 10 minutes. The patient sought medical advice, admitted to Hospital, investigated by chest X-ray, ECG and ECHO and advised to take long acting penicillin for life and sublingual nitrates during attacks of chest pain.
- No syncope
- No symptoms of pulmonary congestion.

❖ **Past history**

- There is past history of **rheumatic fever** since he was 14 years old, manifested by fever and arthritis, investigated by CBC, ESR and ECHO, treated by aspirin and the condition relieved and he was advised to take long acting penicillin for life and it was recurrent several times.
- **20 years ago**, the patient suffered from dizziness and blurring of vision which he sought medical advice, investigated by ECG and X-ray and received treatment in the form of sublingual tablet.
- No DM no HPN.
- No past history of operations or drugs.

❖ **Family history**

- No consanguinity.
- No common diseases in the family.
- No similar condition in the family.

❖ **General exam**

- **Temperature :** 37°
- **Blood pressure :** 110/70
- **Pulse :** regular pulse, 75 /minute, average volume, no special characters, vessel wall is not felt, equal on both sides with intact peripheral pulsations with no radio-femoral delay.

→ **Notice in some cases of AS the pulse is too weak to be felt.**

- Average built.
- No cyanosis, pallor or jaundice.
- No special decubitus (The patient is lying free flat comfortable in bed).
- No oedema L.L
- No clubbing
- Neck veins are pulsating not congested.

❖ **Local exam**

1. **Inspection and palpation .**

- Normal shape of chest.
- No dilated veins.
- No scars of cardiac surgery.
- No pericardial bulge.
- Regarding pulsations :

- ❖ **Apex:** Regular apex, 75/min, lies in left 5th space MCL, localized, heaving in character, with no thrill and no rocking movement

- ❖ Otherwise, apart from weak epigastric pulsations originating from aorta, no other visible or palpable pulsations

2. **By palpation only :**

- **Thrill** over the 1st aortic area, propagated to the neck (In some cases, this thrill is not present).
- **Pulsations** the same as above.
- **Palpable sound:** there is no palpable 1st heart sound or diastolic shock.

3. Percussion :

- **Hepatic dullness** in the Rt. 6th space MCL, no dullness in the Rt. parasternal area, both aortic and pulmonary areas are resonant, preserved waist of the heart, no dullness outside the apex, lower end of the sternum is impaired note with dull bare area.

4. Auscultation

- **2nd sound** muffled.
- Systolic harsh **murmur**, maximum intensity over the 1st aortic area propagated to the neck and the apex increased on setting leaning forward and holding breath in full expiration, it's organic of grade IV/VI with associated thrill (The thrill may not be present and the murmur of grade III/IV).
- **Ejection click** may be heard !!

Discussion :

? **What is your diagnosis?**

☐ Rheumatic heart disease, in the form of isolated aortic stenosis, the patient is compensated and not complicated.

? **Why Rheumatic?** → from history.

? **Why AS?** → from history, exam (Systolic thrill over the 1st aortic area, data of auscultation).

? **Why Compensated?**

☐ From (Absence of symptoms and signs of pulmonary congestion) as No signs of LVF: apical gallop, pulmonary rales, bilateral basal crepitations.

☐ No complication detected

? **What are the causes of AS that you know?**

☐ Revise them from your theoretical book

? **What are the most common causes of AS ?**

☐ Rheumatic Fever, Congenital, Calcific.

? **How could you differentiate between the 3 main causes of AS ?**

	A. Congenital	B. Rheumatic	C. Calcific
○ Age	▪ Young	▪ Middle	▪ Old
○ History of RF	▪ Absent	▪ Present	▪ Absent
○ Type	▪ Valvular or subvalvular or supra-valvular	▪ Valvular	▪ Valvular
○ Associations	▪ Congenital anomalies	▪ Other valvular lesions	▪ Atherosclerosis

? **Which type of the lesion in rheumatic AS, valvular, or subvalvular or supra-valvular?**

☐ Valvular

? **How to prove that is valvular ?**

☐ By presence of Ejection click

? **Why is S2 weak in the patient with AS?**

☐ Due to presence of low pressure over the aortic cusps which will close weakly

? **In which condition is S2 normal in AS ?**

☐ In cases of AS which are associated with AR (double aortic).

? **What are the signs of severity of AS?**

☐ Slow rising pulse

☐ Narrow pulse pressure

☐ Fourth heart sound

☐ Heart failure

☐ History of collapse

? **What additional sound can you hear in AS ?**

☐ Systolic ejection click: due to opening of the rigid cusps.

- ? What do you expect to find in examination of the pulse in AS ?
- ☐ Pulsus parvus et tardus (plateau pulse): rises slowly, of small volume, returns slowly.
- ☐ Pulsus bisferiens: bifid pulse occurring in double aortic lesion with predominant AR.
- ? What can you hear over the mitral area in this patient ?
- ☐ S4 !!
- ☐ Propagated murmur of aortic stenosis.
- ? What can you hear over the pulmonary area in this patient ?
- ☐ Reversed splitting of the second heart sound.
- ? What causes angina in AS ?
- ☐ Reduced coronary blood flow: due to low CO & shortened diastole.
- ☐ Left ventricular hypertrophy: increases the myocardial O₂ demands.
- ☐ Associated coronary atherosclerosis: especially in calcific AS.
- ? What other complications of AS do you know ?
- ☐ Revise them from your theoretical book
- ? What type of heart failure can occur in AS, left-sided or right-sided?
- ☐ Left-sided heart failure due to left ventricular failure & pulmonary congestion.. However, Right-sided heart failure can also occur late secondary to left-sided heart failure.

? How would you proceed to manage this case??

I. Investigations

- ? What do you expect to find in the ECG of a patient with AS ?
- 1. ECG: LVE.
- ? What do you expect to find in the chest x-ray of a patient with AS ?
- 2. CXR for chamber enlargement.
- LVE.
- Post stenotic dilatation
- Pulmonary congestion.
- Calcification.
- ? What is the most important investigation of AS ?
- 3. Echocardiography (investigation of choice, the most diagnostic the most important) to
 - Detects the severity of valve lesion ($< 0.8 \text{ cm}^2 \rightarrow$ severe AS)
 - Chamber enlargement.
- 4. Catheterization:
 - Detects the severity (it can measure the pressure gradient across aortic valve)

II. Treatment

A. Medical treatment

- Beta blockers for angina
- Prophylaxis against IE & rheumatic activity.
- Treatment of complications e.g. HF, AF, infections ...

B. Interventional (ballon aortic valvoplasty).

C. Surgical (valvotomy or valve replacement).

? What are the indications of valve replacement of that patient?

☐ Indications of surgery in AS: (aortic valve replacement):

- A. Presence of symptoms.
- B. Pressure gradient more than 50 mmHg.
- C. Valve area less than 0.8 cm^2 (normally: $3-4 \text{ cm}^2$).

? What are the surface anatomy of aortic valve?

- 3 semilunar cusps attached to a fibrous valve ring .
- In about 1 % of individuals , only 2 cusps are present (Bicuspid aortic valve).
- Surface area is about $3-4 \text{ cm}^2$.

Note

- You may find peripheral signs of aortic regurge with signs of aortic stenosis this mean you are dealing with double aortic lesion.

- ❖ **Personal history:**
- ❖ **Complaint:**
 - **Awareness of heart beats, one week duration.**
- ❖ **History of present illness:**
 - The condition started 10 years ago by gradual onset, progressive course of rapid regular **palpitation** increased with exertion. The patient sought medical advice, investigated by chest X-ray, ECG and ECHO and the patient was advised to be operated but unfortunately, the patient refused the operation.
 - Six years later, the patient developed retrosternal constricting **chest pain** radiated to the left shoulder increased by exertion, relieved by rest and oral treatment and lasted for 5 minutes. The patient sought medical advice, investigated by chest X-ray, ECG and ECHO, treated by sublingual nitrate.
 - The patient was quite well till one week ago when he re-suffered from gradual onset, progressive course of rapid regular **palpitation** increased with exertion.
 - No symptoms of **pulmonary congestion**
 - No symptoms of **low COP**
- ❖ **Past history**
 - There is past history of **rheumatic fever** since he was 10 years old, manifested by fever and arthritis, investigated by CBC, ESR and ECHO, treated by aspirin and the condition relieved and he was advised to take long acting penicillin for life with no recurrence.
 - No DM no HPN.
 - No past history of operations or drugs.
- ❖ **Family history**
 - No consanguinity
 - No common diseases
 - No similar condition
- ❖ **General exam**
 - **Temperature** : 37
 - **Blood pressure** : 150/50
 - **Pulse**: Regular pulse, 75 /minute, big pulse volume, water hammer pulse, vessel wall is not felt, equal on both sides with intact peripheral pulsations, no radio-femoral delay.
 - **Average built.**
 - **No cyanosis, pallor or jaundice.**
 - **No special decubitus** (the patient is lying free flat comfortable in bed).
 - **Head & neck**
 - **Corrigan sign**: strong visible carotid pulsations.
 - **Carotid thrill** (shudder) :systolic thrill.
 - **U.L**
 - **Big pulse volume**
 - **Water hammer pulse**: rapid upstroke, rapid downstroke, big pulse volume
 - **L.L**
 - **Pistol shot** : loud sound with each systole.
- ❖ **Local exam**
- A. Inspection & palpation**
 - Normal shape of chest.
 - No dilated veins.
 - No scars of cardiac surgery.
 - No pericardial bulge.
 - Regarding pulsations :
- ❖ **Apex**: Regular apex, 75/min, lies in left 5th space MCL, localized, hyperdynamic in character, thrill and no rocking movement.
- ❖ Otherwise, apart from epigastric pulsations originating from aorta no other visible or palpable pulsations.

B. By palpation only:

- **Pulsations** the same as above.
- **Palpable sound** : there is no palpable 1st heart sound or diastolic shock.
- **No thrill** either on base, left parasternal area or on apex.

C. Percussion :

- **Hepatic dullness** in the Rt. 5th space MCL, no dullness in the Rt. parasternal area, both aortic and pulmonary areas are resonant, preserved waist of the heart, no dullness outside the apex, lower end of the sternum is impaired note with dull bare area.

D. Auscultation:

- **2nd sound** normal or muffled.
- Diastolic soft blowing **murmur**, maximum intensity over the 2nd aortic area propagated to the apex, increased on setting, leaning forward and holding breath in full expiration, it's organic, grade III/VI with no thrill.

❖ NB :

- **In AR:** notice you can hear systolic soft murmur localized over the 1st aortic area with no associated thrill (**functional** murmur of relative aortic stenosis).
- To be differentiated from double aortic lesion in which the murmur is propagated to the neck and the apex, associated with thrill and the patient complains of low COP symptoms.

◦ Discussion :**? What is your diagnosis ?**

- 📖 **Rheumatic heart disease**, in the form of isolated aortic regurge, the patient is compensated and not complicated.

? Why Rheumatic? → from history.**? Why AR? → from history, exam (peripheral signs of AR, hyperdynamic apex epigastric pulsation from aorta and data of auscultation may help you).****? Why Compensated? → from (absence of symptoms and signs of pulmonary congestion) as no signs of LVF : apical gallop, pulsus alternans, bilateral basal crepitations.**

→ No Complication detected

? What are the other causes of AR that you know?

- 📖 Revise them from your theoretical book

? Is it regular or irregular palpitation?

- 📖 Regular palpitation, because it is due to increased force of cardiac contraction.

? So, what does it mean if the patient with AR complains of irregular palpitation?

- 📖 It means that his case got complicated by an arrhythmia which made the palpitation become irregular.

? What causes angina in AR ?

- 📖 Two types of angina occur in aortic regurge "

- Classic angina of effort: due to decreased diastolic blood pressure due to regurge.
- Angina of Lewis: nocturnal & associated with autonomic disturbances such as sweating & tachycardia.

? What are the other causes of systolic thrill over the carotids ?

- 📖 See general chapter

? What can you hear over the mitral area in this patient ?

1. S₃.
2. Propagated murmur of aortic regurge.
3. Pan systolic murmur of functional mitral regurge.
4. Austin Flint murmur (mid-diastolic) due to:
 - Elevation of anterior leaflet of the mitral valve by the regurgitant blood from the aorta causes relative MS.

? What are the other signs of AR that you know (peripheral signs of AR)?

- 📖 See before

? Where else can you search for capillary pulsations ?

- 📖 See before

? What are the expected complications of AR ?

- 📖 Revise them from your theoretical book

- ? What type of heart failure can occur in AR, left-sided or right-sided?
 Left-sided heart failure due to left ventricular failure & pulmonary congestion.. However, Right-sided heart failure can also occur late secondary to left-sided heart failure.
 ? What are the Differential diagnosis of murmurs with AR?
 Relative AS: due to increase in volume of blood pumped in systole
 Relative MS: as regurgitant blood interferes with anterior mitral leaflet (Austin flint)
 Relative MR: secondary to LV dilatation

? How would you proceed to manage this case?

I. Investigations

? What do you expect to find in the ECG of a patient with AS ?

1. ECG: LVE.

? What do you expect to find in the chest x-ray of a patient with AS ?

2. CXR for chamber enlargement.

- LVE & dilated aorta (boot-shaped heart)

? What is the most important investigation of AS ?

3. Echocardiography (investigation of choice, the most diagnostic, the most important) to

- LVE

- Detects the severity of valve lesion

4. Catheterization:

- LVE & Detects the severity of the lesion

II. Treatment

D. Medical treatment

- Prophylaxis against IE & rheumatic activity.
- Treatment of complications e.g. HF , AF , infections ...

E. Surgical (valve replacement).

? When would you ttt this patient surgically ?

Indications of surgery in AR: (aortic valve replacement):

- Presence of symptoms.
- Progressive cardiomegaly.
- LV dysfunction.

M.S. with T.R.

❖ **Personal history**❖ **Complaint :**

- **Shortness of breath, 4 month duration**

❖ **History of present illness:**

- ❖ The condition started 10 years ago by gradual onset and progressive course of **dyspnea**. The patient experienced exertional dyspnea on less than ordinary effort ,on climbing the 1st floor not associated with orthopnea or PND. this dyspnea was associated with gradual onset ,progressive course rapid irregular **palpitation** increased with exertion The patient sought medical advice, Investigated by chest X-ray ,ECG and ECHO diagnosed as tight mitral stenosis and treated by mitral valve replacement
- ❖ The patient was quite well, after operation, till 4 months ago when he redeveloped again exertional **dyspnea** on less than ordinary effort associated with **orthopnea**, the patient used to sleep on three pillows and **PND**, after one or two hours of sleep the patient usually wakes up with dyspnea ,cough and wheeze then after 15 minutes the condition usually relieves . this dyspnea is associated with gradual onset, progressive course rapid irregular **palpitation** increased with exertion. Two months later, the patient experienced **dyspepsia** ,**dull aching pain in the right upper abdomen** associated with bilateral pitting painless **lower limb edema** extending to thigh level and also gradual onset ,progressive course of generalized **abdominal distension**.

❖ **Past history:**

- There is past history of **rheumatic fever** since he was 14 years old, manifested by fever and arthritis, investigated by CBC,ESR and ECHO ,treated by aspirin and the condition relieved and he was advised to take long acting penicillin for life and it was recurrent several times
- No DM no HTN
- No past history of operations or drugs

❖ **Family history:**

- No consanguinity , No common diseases , No similar condition

❖ **General exam****A. Overview**

- **Temperature** : 37
- **Blood pressure** 110/70
- **Pulse** : markedly irregular pulse ,75 /minute, variable volume , pulsus deficit >10,vessel wall is not felt ,equal on both sides with intact peripheral pulsations
- **Orthopnea**
- Average **built**, no **pallor** no **jaundice** ,no **cyanosis**

B. Head & neck

- **Congested pulsating neck veins** extending to lobule of the ear , congestion decreased by inspiration with +ve hepato-jugular reflux. There is systolic expansion with absent a wave
- No malar flush

C. Extremities

- Bilateral pitting **lower limb edema** extending to mid thigh level and not tender

❖ **Local exam :****A. Inspection and palpation**

- **Barrel shaped chest** "*just association in the case*"
- **Median sternotomy scar** of valve replacement surgery.
- **Pericardial bulge**.
- No dilated veins.

❖ **Regarding pulsations :**

- **Apex**: markedly irregular apex ,90/min, lies in left 5th space outside MCL, diffuse , slappy in character ,with no thrill and no rocking movement .
- **Pulmonary pulsation** detected by palpation by tips of fingers.
- **Left parasternal pulsation** detected by palpation by base of the hand , increased by chest deflation.
- **Epigastric pulsation** detected by both inspection and palpation by tips of fingers increased by deep inspiration.
- There is **hepatic pulsation** detected by bimanual palpation of the liver

B. By palpation only :

- **Pulsations** the same as above
- **Palpable sound** : there is palpable 1st heart sound (slappy apex), but no diastolic shock "may be present in such cases, in our case may be masked by hyperinflation"
- No **thrill** either on apex ,left parasternal area or on base

C. Percussion :

- **Hepatic dullness** in the Rt 5th space MCL ,there is dullness in the Rt parasternal area , Aortic area is resonant while there is **3 finger dullness in pulmonary area, obliterated waist** of the heart(4 finger dullness in left 3rd space ,normally just 2 finger dullness),no dullness outside the apex ,**lower end of the sternum is stony dull with dull bare area**

D. Auscultation

1. Over the apex

- **1st sound** accentuated
- Diastolic rumbling **murmur** ,localized over the apex ,increased on lying in the left lateral position ,it's organic ,grade III/VI with no thrill
- **OS** may be heard !!

❖ NB :

- **In AF** → notice the following difference: **Variable 1st heart sound with no presystolic accentuation of the murmur**

2. Over the pulmonary area

- **2nd sound** accentuated
- Ejection systolic murmur of relative pulmonary stenosis (systolic, localized, soft, no thrill, grade III/IV, increased with setting ,leaning forward) .

3. Over the tricuspid area

- Pan-systolic murmur of functional tricuspid regurge (systolic ,localized, soft ,no thrill, grade III/IV ,increased with inspiration "carvallo's sign")

❖ Examination of other systems

- In this case ,you should search for **other signs of systemic congestion** other than edema and neck veins like ascites and enlarged tender liver as a part of abdominal examination in such case

❖ Discussion

? What is your Diagnosis ?

📖 **Rheumatic heart disease , in the form of mitral restenosis with functional tricuspid regurge ,the patient is decompensated and complicated by AF**

Or

📖 **Rheumatic heart disease , in the form of mitral restenosis regurge ,the patient is decompensated and complicated by AF and functional tricuspid regurge**

? Why Rheumatic ? → from history

? Why MS? → from history , exam (irregular pulse ,slappy apex, auscultation,investigation)

? Why TR? → from history ,general signs of systemic congestion, signs of right ventricular enlargement, auscultation and investigation)

? Why Decompensated? → from (symptoms and signs of systemic congestion)

? Why Complicated? → by AF (irregular pulse with marked irregularity and pulsus deficit >10)

? Investigations:

- ECG for chamber enlargement and arrhythmia
- CXR for chamber enlargement (plain and contrast)
- ECHO(investigation of choice): for anatomical functional aetiological diagnosis and for detection of complication

? Treatment:

- Medical (prophylactic and symptomatic)
- Surgical (mitral valve replacement "2nd time for our case")

Double Mitral Lesion

❖ Personal history:

❖ Complaint :

- Palpitation of 10 days duration.

❖ History of present illness:

- o The condition started 3 years duration by rapid **regular palpitation**. The palpitation increased by exertion & stress. It is relieved by rest. 2 years ago, the patient experienced **exertional dyspnea** on less than ordinary effort, on climbing the 1st floor not associated with orthopnea or PND.
- o The patient remained symptom-free then he developed **rapid irregular palpitation** increased by exercise & relieved by rest. He experienced orthopnea & PND. He gave a history of symptoms of systemic congestion as **LL edema** 6 months ago of progressive course associated with **dull aching pain in RT hypochondrium** increasing with exertion relieving by rest.
- o No symptoms of low COP.
- o No history neither of chest pain nor of pressure symptoms.

❖ Past history:

- o There is past history of **rheumatic fever** Since he was 15 years old, manifested by fever and carditis, investigated by CBC, ESR and ECHO, treated by some medications -not known by the patient- and the condition relieved and he was advised to take long acting penicillin for life and it was recurrent several times.
- o No DM no HPN.
- o Past history of tonsillectomy at the age of 15 years
- o No history of drug allergy

❖ Family history

- o +ve consanguinity.
- o No common diseases in the family, No similar condition in the family.

❖ General exam

- o **Temperature:** 37°
- o **Blood pressure:** 110/70.
- o **Pulse:** irregular pulse, 75/minute, equal volume on both side, pulsus deficit>10, vessel wall is not felt, equal on both sides with intact peripheral pulsation with radio-femoral delay.
- o **RR :** 22 \ min
- o **Mentality:** The patient is fully conscious oriented to time, place & persons. He is of average mood & affect & of good memory & average intelligence. He is co-operative.
- o **Average built.**
- o **No cyanosis, pallor or jaundice.**
- o The patient is **orthopneic**.
- o **Bilateral pitting L.L edema** up to mid leg.
- o **No clubbing.**
- o **Neck veins:** congested pulsating neck veins, Systolic expansion & absent a wave.

❖ Systemic overview

- o **Chest:** fine bilateral basal crepitations.
- o **Liver:** enlarged tender with systolic expansion.

❖ Local exam:

1. Inspection and palpation

- o Normal shape of chest.
- o No dilated veins on chest wall.
- o No pericardial bulge.
- o Regarding pulsations :
- o **Apex:** Irregular apex, 90/min, lies in left 6th space outside MCL, diffuse, hyperdynamic in character, with systolic thrill in left lateral position.
- **Weak epigastric pulsations** originating from above & from RT side (of hepatic origin),
- **RT & Lt parasternal pulsations.**
- o No other visible or palpable pulsations.

2. Percussion :

- **Hepatic dullness** in the Right 5th space MCL, dullness in the right parasternal area & lower third of sternum, increased dullness over bare area, both aortic and pulmonary areas are resonant, preserved waist of the heart, dullness outside the apex.

3. Auscultation

A. Over the apex:

- Variable S1
- S3 sound !!
- Pansystolic soft murmur heard maximally over the apex, propagated laterally to the axilla & upwards to the base of the heart, heard best in the left lateral position & with exercise.

B. Over tricuspid area: Pansystolic soft murmur which increases by inspiration

❖ Discussion :

? What is your diagnosis?

📖 Rheumatic heart disease, Mitral valve disease, double mitral, TR with biventricular enlargement. non Compensated, Complicated by AF.

- **Why Rheumatic?** → from history & presence of multi valve lesions
- **Why double mitral?** →

1. Because there is clinical evidence of MR in the form of:	2. Because there is clinical evidence of MS in the form of:
❖ From history	
○ Palpitation	○ Dyspnea with PND.
❖ From precordial examination	
○ Signs of LV enlargement : Hyperdynamic apex , Localized apex with overlying systolic thrill	○ Signs of RV enlargement: left parasternal uplift, epigastric pulsations, dullness over lower sternum
❖ From auscultation	
↳ S3 sound (protodiastolic) due to volume overload. ↳ Typical murmur of MR: ○ Timing: pansystolic. ○ Character: soft or harsh. ○ Site: over the apex. ○ Propagation: <i>laterally to the axilla</i> (usually), <i>upwards to the base of the heart</i> (uncommonly). ○ Position: heard best in the left lateral position.	↳ Normal S1 over the apex (instead of muffled S1) indirect evidence of the presence of MS. ↳ Typical murmur of MS: was not clear. ↳ Evidence of PH: accentuated S2 over the pulmonary area.

? Why TR ?

📖 From history: Symptoms or RVE (LL edema , RT hypochondrial pain

📖 Examination:

- General : congested pulsating neck veins with systolic expansion & tender pulsating liver
- Local : Signs of RV enlargement
 - By inspection & palpation: Rt parasternal pulsations, epigastric pulsations, diffuse apex
 - By auscultation: pansystolic soft murmur on tricuspid area increased by inspiration.

? **Why Non-Compensated?** → from (presence of symptoms and signs of LVF & RVF).

? **What is the evidence of LVF ?** S3 , fine basal crepitation & orthopnea.

? **What is the main presenting symptom of MR?** Palpitation

? **Is it regular or irregular palpitation of MR?** Regular because it is due to increased force of cardiac contraction

? When does the patient say it is irregular palpitation? If complicated by an arrhythmia: AF, which made the palpitation become irregular

? What proves that the patient developed AF as a complication?

From History

Palpitation changed from regular to irregular.

From General Examination

Systolic expansion in neck veins & absent a wave.

Marked irregularity in pulse rhythm, with pulsus deficit more than 10.

From auscultation: Variable first heart sound

? But, I can hear a mid diastolic rumbling murmur over the apex that you did not mention, what does it indicate?

It indicates the presence of a relative mitral stenosis (not organic) due to increased blood flow across the mitral valve in cases of MR.

? How would you confirm the diagnosis of AF by one single investigation?

ECG will reveal: 1) Irregular rhythm. + 2) Absent p wave.

? Why is S1 muffled in the patient with MR?

Due to failure of proper mitral closure.

? When can you hear a normal or an accentuated S1 in cases of MR?

In cases of MR associated with Mitral Stenosis, i.e. Double Mitral lesion.

? Why is S1 accentuated in the patient with MS?

- Fibrosis of the mitral cusps.

- Forcible closure of the mitral cusps: because:

- They are displaced downwards due to high LA pressure.

- The mitral valve is opened as wide as possible during the diastole.

? In which condition does the murmur of MR propagate upwards to the base of the heart?

In cases where MR is due to disease of the posterior leaflet.

? Can the murmur of MR propagate medially to the tricuspid area?

Sometimes, yes, also in disease of the posterior leaflet.

? On the tricuspid area, how would you differentiate clinically between the pansystolic murmur of propagated MR & the pansystolic murmur of TR?

❖ MR	❖ TR
○ Pansystolic murmur on apex radiating to axilla	○ Pansystolic murmur on tricuspid radiating to apex
○ Inspiration has no effect on murmur	○ Inspiration increase the murmur (caravallo's sign).
○ Associated with ↓ S1, S3 & LVH	○ RVH and pulsating liver & congested neck veins

? How would you proceed to manage this case?

- I would ask for *echocardiography* to prove my diagnosis of Double mitral lesion.

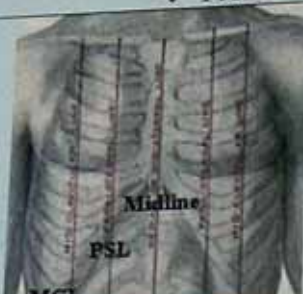
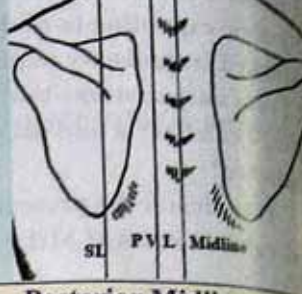
- I would then start treatment which could be: medical or surgical

? What kind of surgery would you do for this patient with double mitral valve?

- Valve replacement.

CHEST

❖ Lines of the chest :

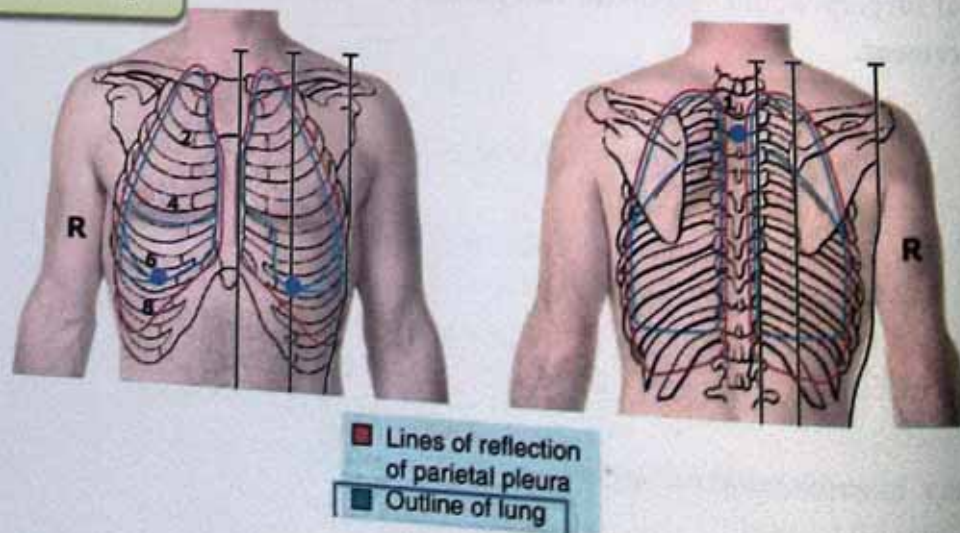
❖ Front	❖ Lateral	❖ Back
 - Anterior Midline : middle of the sternum - Parasternal line (PSL): midway between MCL & lateral border of the sternum - Midclavicular line (MCL) : midway between acromion process and sternoclavicular joint	 - Anterior axillary line (AAL): anterior fold of axilla - Mid axillary line (MAL): apex of axilla - Posterior axillary line (PAL): posterior fold of axilla	 - Posterior Midline : spinous of vertebrae - Paravertebral line (PVL): midway between midline & medial aspect of scapula - Scapular line (SL) : from the angle of scapula

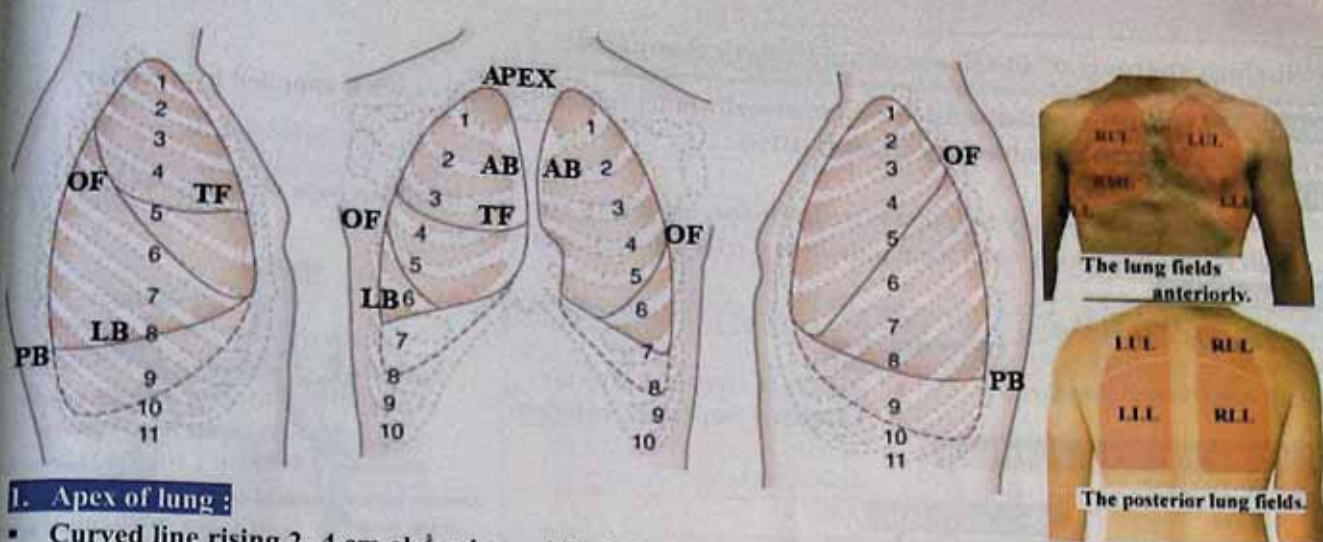
❖ Regions of the chest :

❖ The chest has right & left side, each side has:

1) Anterior aspect	2) Lateral aspect	3) Posterior aspect
 - Supra-mammary area - Mammary area - Infra-mammary area	 - Upper axillary area - Lower axillary area	 - Supra-scapular area - Inter-scapular area - Infra-scapular area

❖ Surface anatomy of the lung:





1. Apex of lung :

- Curved line rising 2- 4 cm above inner 1/3 of clavicle, passing downwards to sternoclavicular joint

2. Anterior border:

- From sternoclavicular joint, line passes downwards & medial to reach middle line at angle of Lewis
- It then descends in midline :

A. **In right lung**, it reaches level of 6th costochondral cartilage

B. **In left lung**, it reaches level of 4th costal cartilage & then deviates for 1 inch to left of sternum (bare are of heart – cardiac notch), then it curves down & medially to end at 6th costochondral cartilage

3. Lower border of lung & pleura:

	MCL	MAL	Scapular line
Upper border of Liver (right side)	5 th space	7 th space	9 th space
Lower border of Lung	6 th rib	8 th rib	10 th rib
Lower border of Pleura	8 th rib	10 th rib	12 th rib

4. Posterior border:

- Runs vertically upwards along sides of vertebral column starting from level of T10 spine below, ending at apex above.

5. Oblique fissure in both lungs :

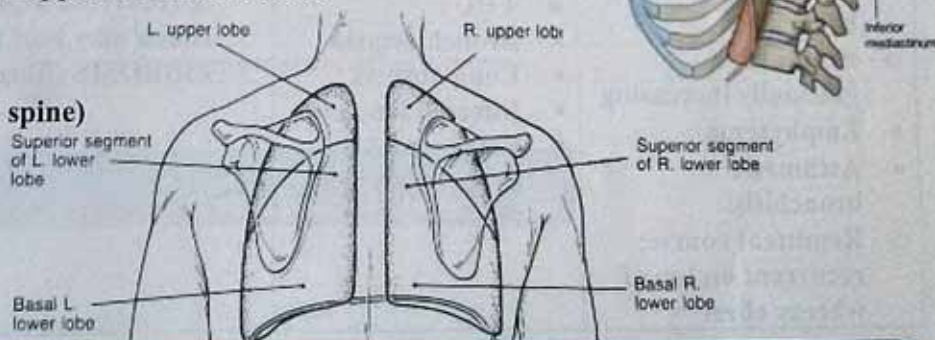
- Line passing down & outwards oblique line from T2 spine, to the 5th rib in mid-axillary line, then to the 6th costochondral cartilage.

6. Transverse fissure of right lung:

- From the 4th costochondral cartilage to meet the line of oblique fissure in MAL
- It divides the upper right lobe into right upper and middle lobes.

7. Bifurcation of trachea :

- Anteriorly at the level of sternal angle,
- Posteriorly at the body of T4 (tip of T2 spine)



- When you examine back, you examine lower lobe mainly
→ When you examine interscapular region, you examine apical segments of lower lobes
- When you examine the front, you examine upper lobe in the left lung and upper and middle lobes in right lung
- When you want to examine middle lobe only, middle lobe of the lung
→ you examine the area between 4th to 6th ribs at MCL line on the right side

❖ Surface anatomy of the Bronchopulmonary Segments:

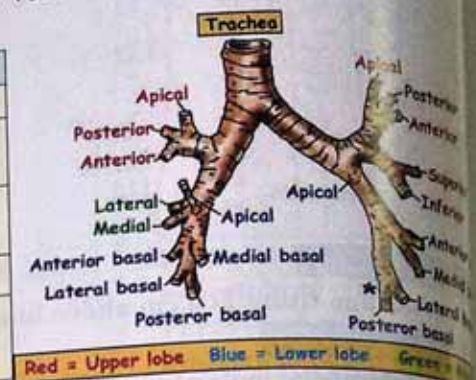
A. Definition : each lung is subdivided into a functionally independent segments each is supplied by a tertiary bronchus called bronchopulmonary segment (BPS)

B. Shape & structure :

- Each Bronchopulmonary Segment is wedge shape, having an apex at hilum & a base at surface of lung
- Each is supplied by a branch from pulmonary artery & pulmonary vein

C. Pattern of BPS :

A. Right lung	B. Left lung
❖ Upper lobe:	
▪ Apical, Anterior, Posterior	▪ Apical, Anterior, Posterior
	▪ Lingular: Superior, Inferior
❖ Middle lobe only in the right lung :	
▪ Medial & Lateral	
❖ Lower lobe:	
▪ Apical	
▪ Anterior basal, Posterior basal, Medial basal, Lateral basal	



CHEST CASES

❖ Case		D. Pulmonary fibrosis	
A. COPD = COLD = COAD	B. Suppurative lung syndrome = cavitary syndrome	C. Pleural effusion	
❖ Patient complains:			
1. Exertional dyspnea : gradual and progressive 2. Cough 3. Expectoration : whitish, moderate amount, odourless 4. ± Wheezes	1. Cough & expectoration : ⚰ Big amount ⚰ Bad odour "foetid" ⚰ Purulent "yellowish or greenish" ⚰ Postural 2. Dyspnea 3. ± wheezes 4. ± hemoptysis	1. Pleuritic pain, character : ○ Stitching ○ Superficial ○ Localized may be referred ○ Decrease by shallow breathing & lying on diseased side ○ Increased by deep breathing, cough or sneezing 2. Dry cough 3. Dyspnea : acute & at rest 4. History of pleural effusion & recurrent aspiration which may lead to <i>pleural FIBROSIS</i> (fibrothorax)	1. dyspnea : exertional, gradually, progressive 2. DRY cough 3. Dull aching Chest pain
❖ Due to:	❖ N.B.:		❖ N.B.:
1. Reversible : Bronchial asthma 2. Irreversible : • Chronic obstructive bronchitis: ○ Progressive gradually increasing • Emphysema • Asthmatic bronchitis: ○ Remittent course; recurrent on top of wheezy chest	4 DD : ▪ Bronchiectasis ▪ Lung abscess ▪ Infected lung cyst ▪ Empyema & broncho-pleural fistula		Types of pulmonary fibrosis:
			A. Parenchymatous fibrosis: • Occurs as a complication of various lung diseases e.g. ○ TB (most of cases) ○ Lung abscess ○ Bronchiectasis B. Interstitial fibrosis: idiopathic pulmonary fibrosis (Hamman Rich syndrome)

⚡ N.B.:

A. Definition of Chronic bronchitis: productive cough every day or most of days + duration ≥ 3 months/year 2 successive years

B. Asthmatic bronchitis: = BA + Chronic bronchitis

C. TB can cause :

- TB cavity {SLS (Lung abscess) + TB} → may cause fibrosis
- Bronchiectasis → may cause fibrosis
- Pleural effusion → may cause fibrosis
- Parenchymatous fibrosis

CHEST HISTORY

1) Personal History

1. <u>Name</u> : الإسم الثلاثي	2. <u>Age</u> : ○ Young : TB, adenoma ○ Old : bronchial carcinoma	3. <u>Sex</u> : ○ Females : adenoma ○ Males : Carcinoma
4. <u>Occupation</u> : ○ Dusty work: emphysema, pneumoconiosis ○ Farmers: bronchial asthma, bilharzial cor pulmonale ○ Asbestosis : mesothelioma	5. <u>Marriage</u> : for TB transmission to the whole family members , ask about fertility because infertility associated with Kartagener's syndrome 6. <u>Menstrual history</u> if female.	7. <u>Residence & address</u> : chronic bronchitis is more common in areas of high pollution
8. <u>Habits of medical importance</u> : 1. Cigarette smoking : bronchial carcinoma, chronic bronchitis & COPD 2. Goza : transmission of infection, COPD 3. IV addicts : fungal & staph acute bacterial endocarditis	9. <u>Handedness</u> .	

2) Complaint + duration: أي اللي جابك المستشفى ؟

❖ Common complaints :

▪ Shortness of breath.	▪ Cough	▪ Excessive spitting	▪ Coughing of blood	▪ Chest pain
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3) History of present illness = HPI المرض بدأ معاك إزاي , آخر مرة كنت كويس فيها إمتى ؟

1. Symptoms of <u>C</u> HEST: ▪ C: Cough ▪ H: Hemoptysis ▪ E: Expectoration ▪ S: Shortness Of Breath ▪ T: Wheezes (تزييق Tazee2)	2. Symptoms of systemic <u>C</u> ongestion: <u>C</u> or-pulmonale ▪ Cor-pulmonale: right ventricular enlargement ± failure secondary to chest disease 3. <u>C</u> yanosis : Suggestive Symptoms of respiratory failure 4. <u>C</u> onstitutional (toxic) symptoms
5. <u>P</u> leural effusion aspiration	
6. Chest <u>P</u> ain	
7. <u>P</u> ressure symptom	
8. Investigations & treatment received	9. Symptoms of other systems

تتكتب في ال complaint بكلام العيان
تتكتب في H/O of present illness عادي

1. Symptoms of CHEST

I) Cough :

➤ Cough

A. **OCD, ↑, ↓ (ATP)**

في كحه ؟ ظهرت فجأة ولا ازاي ؟ ايه اللي بيوزدها وايه اللي بيقلها ؟

C. **Time :** ؟ بتزيد في وقت معين ؟ بتزيد ليل ولا الصبح ولا ثابتة ؟

- 1 – 2 hours after sleep i.e. nocturnal (cardiac asthma)
- Early in the morning (Bronchiectasis & Bronchial Asthma; BA)

E. **Relation to Position (SLS)** ؟ هل فيه وضع معين بيوزد الكحة ؟ هل الكحة ببلغم ولا ناشفة ؟

B. **Associated symptoms** ؟

- Dyspnea & wheezes in COPD
- Toxemia in Suppurative Lung Syndrome (SLS)

D. **Type :**

هل الكحة ديه مستمرة طول الوقت ولا في صورة نوبات بتيجي وتروح ؟

- Continuous
- Paroxysmal (BA, SLS)

F. **Dry** ناشفة or **Productive** ببلغم

➤ If productive = **Expectoration**; analyze (**APCD +SO**) :

➤ Excessive spitting

➤ **Amount:** ؟ كمية البلغم اداي في اليوم ؟ Cup = 200 cc /day

- SLS : big, thick
- COPD: moderate amount
- BA: small amount, viscid pellets

➤ **Associated symptoms** ؟ هل البلغم ده بيوزد في وضع معين ؟ e.g. wheezes (COPD), toxemia (SLS)

➤ **Postural variation is characteristic to SLS:** ؟

- Lung abscess: ↑ by lying on healthy side
- Bronchiectasis : ↑ by stooping (leaning) forward

➤ **Color & Consistency (Character):** ؟ لون البلغم ايه ؟ ابيض ولا ليه لون ؟

	Color	Consistency (Character)
	Whitish	Mucoid
COPD		
COPD with secondary infection	○ Greenish (anaerobic infection) ○ Yellowish (pyogenic infection)	Muco-purulent
SLS	Yellowish	Purulent

➤ **Diurnal variation (time):** ؟ البلغم ده بيوزد في النهار ولا ليل ؟

- Nocturnal: cardiac
- Early in the morning : chest : Bronchiectasis & Bronchial Asthma (BA)

➤ **Seasonal Variation :** ؟ صيف ولا شتا ؟

- Allergy (pollen in spring) : allergic rhinitis & Bronchial asthma
- Bronchiectasis : more in winter

➤ **Odor :** ؟ هل البلغم له رائحة ؟

- Odourless
- Offensive = foetid = foul sputum in SLS or infection by anaerobes

2) Hemoptysis : in TB & SLS especially bronchiectasis

➤ Coughing of blood = blood spitting

متج دم ؟ من إمتة ؟ وظهرت بالتدريج ولا فجأة ؟ كام مرة كحيت دم ؟

النية اداي ؟ لونه ايه ؟ الدم اللي كحيتة كان دم بس ولا كان دم ومعاها حاجه ؟

الوقت الوعي ؟ هل نزفت من حته تاتيه ؟ هل احتجت ينقلوك للمستشفى ؟ احتجت ينقلوك دم ؟

▪ **OCD, ↑, ↓, then analysis (ABCD) :**

A. **Attack :** number, date, before the attack..., during the attack..., after the attack

B. **Blood :** amount, content, color

	❖ Hemoptysis	❖ Hematemesis
▪ Before attack:	Cough	Nausea, vomiting
▪ During attack:		
○ Symptoms	Coughing blood	Vomiting blood
○ Colour	Bright red blood	Dark; Coffee-ground (hematine) المصروق
○ Content	Mixed with air (frothy)	Mixed with food
○ Chemical reaction (pH)	Alkaline	Acidic
▪ After attack	Blood tinged sputum for a while	Melena and constipation
▪ Examination	Chest , CVS signs	GIT signs

C. Circulatory compensation : hospital admission, blood transfusion D. Dysfunction : Disturbed conscious level, bleeding from other orifices ... years ago, the patient had {number of attacks....} of hemoptysis of acute onset, {...} course, the attack was in {Date e.g. The fourth of April, nineteen ninety three. (Apr. 1993)} ...was preceded few days before by cough & expectoration of {Amount e.g. small or moderate or large amount} of {blood tinged sputum in bronchiectasis or frank blood in bronchiectasis sicca haemorrhagica} bright red in colour, containing froth then followed by blood tinged sputum for a ... days, {±} associated with loss of consciousness, the patient sought medical advice, he was admitted to ... hospital, investigated by ..., received treatment in the form of	
3) Dyspnea: هل عندك كرشة نفس، نهجان؟ Shortness of breath, for analysis : see cardiology	
4) Wheezes: (Tazee2 تزييق) : عندك تزييق في صدرك ؟ Definition: musical sound produced by passage of air through narrow bronchi; type : If the patient in-between attacks: - Still have wheezes i.e. Continuous i.e. irreversible : asthmatic bronchitis - Free i.e. Paroxysmal i.e. reversible : BA; ask about time of attack, ↑, ↓	
2. Symptoms of systemic Congestion indicating right side heart failure (cor-pulmonale) رجلك أو بطنك نفخت ؟ في وجع في جنبك اليمين ؟ عينك اصفرت ؟ - Lower limb edema (LL swelling) - Ascites (abdominal distension) - Hepatic congestion: - Jaundice - Pain in Right Hypochondrium - GIT congestion:(nausea , vomiting, etc...) For analysis of each one; see cardiology.	3. Cyanosis ازرقيت ؟ شفائيك بتزرق ؟ ضوافرك بتزرق ؟ - OCD, ↑, ↓, Relation to position or exertion. - Cyanosis is a suggestive symptom of respiratory failure; respiratory failure is a laboratory diagnosis ; ABG. ± Interstitial pulmonary fibrosis
4. Constitutional (toxic) symptoms عندك فقدان للشهية ؟ وزنك خس ؟ بتعرق كثير ؟ - Night fever , night sweats, loss of weight & loss of appetite - Suggestive of malignancy, TB toxemia & SLS e.g. lung abscess	5. Pleural effusion aspiration, Analysis: هل عملت بزل أو تشفيط ؟ - Amount : if 1 bottle (500 cc) ؟ - Aspect : clear or turbid ؟ كان صافي ولا معكر ؟ - Colour ؟ كان لونه اي ؟ - Complicated or not (e.g. infection, fistula) - هل حصلك اي مشاكل زي التهابات او فتحة على الجلد مرضتش تقفل ؟ - Recurred or not ؟ هل خفيت ولا الموضوع اكرر معاك تاني ؟
6. Pressure symptoms for mediastinal syndrome هل صوتك انتبح ؟ هل عندك صعوبة في البلع ؟ For analysis; see cardiology	7. Chest Pain هل عندك ألم في صدرك ؟ For analysis; see cardiology.
8. Investigation & treatment: عملت فحوصات اي ؟ واخذت علاج اي ؟ Chest investigations as : - Sputum examination, Culture & sensitivity of expectorated sputum - Tuberculin skin test, Chest X-ray, CT chest - Arterial blood gases (ABG) Treatment received as : - Bronchodilators, Steroids, Antibiotics, Aminophylline - Nebulization & Domestic o2 therapy	9. Other systems Symptoms of other systems e.g. CVS, GIT, neurology هل عندك مشاكل تانية في جسمك ؟ وتاخذ كل سيستم وتسال العيان على الأعراض الكبيرة اللي فيه بسرعة اوي

4) Past history as usual but focus on tuberculosis:

- جالك زمان كحة ودم وبلغم ؟ كنت بتسخن بليل وتبل الملايه عرق ؟ اتحجزت في مستشفى الصدر وأخذت علاج لفترة طويلة ؟
- Constitutional (Toxic) symptoms e.g. Night fever , night sweats, loss of weight & loss of appetite
 - Chest symptoms e.g. cough, expectoration , dyspnea, hemoptysis
 - Admission to chest sanatorium, Antituberculous drugs intake
 - Multiple drugs for a long period (minimum 6 months)
 - Repeated daily intramuscular injection with streptomycin or oral intake of rifampicin with red discoloration of urine

- Start the history of the patient by TB at the beginning of HPI
- Don't forget to ask about DM in the past history, if positive analyze and start HPI with the patient is known to diabetic

لا بد أن تذكر ال DM سواء كان +ve أو -ve لأن TB follows DM like its shadow!

5) Family history as usual & asked for allergy, Bronchial Asthma, TB

Algorithm of Chest History For Diagnosis

Ask about the time of chest symptoms; cough, dyspnea, wheezes

Paroxysmal:
Bronchial
asthma

Continuous;
Then ask about expectoration

YES, then ask about:
Amount, Posture, Colour, Odour

NO expectoration:
Fibrosis due to:

- TB or
- Recurrent aspiration of pleural effusion

- Small to moderate amount
- Odourless
- Whitish mucoid sputum
- No relation to position

COPD

- Big amount
- Bad odour "foetid"
- Purulent "yellowish or greenish"
- Postural variation

Suppurative lung syndromes
Then ask about special posture

○ Lung abscess:
↑ By lying on one side (healthy side)

○ Bronchiectasis:
↑ By stooping (leaning) forward

General Examination Of Chest Case

❖ Examination as general examination but stress on the following:

I. Vital signs

❖ Temperature	❖ Blood pressure	❖ Respiratory Movements
- High grade fever : pneumonia, lung abscess - Low grade fever : TB, bronchial carcinoma	- Hypertension : polyarteritis nodosa causing bronchial asthma - Hypotension : - Right side heart failure in cor-pulmonale, - Miliary TB causing Addison's disease	See details later in the chapter.
❖ Pulse: مهم جدا جدا جدا - Rate: - Tachycardia: due to hypoxia & respiratory distress & B stimulants in treatment of BA - Bradycardia: sarcoidosis infiltrating AV bundle - Rhythm : - Irregular : multifocal atrial tachycardia (MAT) in chronic hypoxia (e.g. COPD) or atrial fibrillation (AF) - Volume : - Big pulse volume : VD due to hypoxia - Small pulse volume: ↓ COP due to cor-pulmonale (P.HTN & RV failure) - Special Character: - Water hammer pulse : emphysema (VD caused by hypoxia) - Pulsus paradoxus in severe COPD or status asthmaticus - Unequal pulse volume : thoracic inlet syndrome		

II. General overview

A. Appearance	B. Built	C. Consciousness	C. Colors	D. Decubitus	E. Expression
- Toxic in TB, suppurative lung syndrome - Cachectic if malignancy	- Underbuilt : TB since childhood - Overbuilt: Pickwickian syndrome with corpulmonale	Cloudy consciousness in respiratory failure	See below	- Orthopnea : in severe emphysema, ascites in cor-pulmonale - lateral decubitus : lung abscess (on healthy side) & pleurisy (on diseased side)	Moon face due to steroids therapy for bronchial asthma

❖ What are the Causes of ... in chest case ?

❖ Jaundice	❖ Cyanosis	❖ Pallor
1. Hepatocellular: - Cor pulmonale : liver congestion - Rifampicin (anti TB) - Associated viral hepatitis 2. Hemolytic: pulmonary embolism 3. Obstructive: bronchial carcinoma (liver metastases)	- Peripheral: - Right sided Heart Failure (Bilharzial corpulmonale) - SVC thrombosis - Central: - COPD (hypoxic cor pulmonale) - Pulmonary fibrosis, - Pulmonary embolism, - Acute pulmonary failure. - Both central & peripheral: - Massive pulmonary embolism	- Infection - Lung abscess - Bronchiectasis - Pneumonia - Pulmonary embolism - TB - Malignancy

III. Systemic overview :

❖ Head	❖ Neck
1) Eyes: - Argyl Robertson pupil in syphilis with pulmonary hypertension - Puffy lids : chronic cough, cor pulmonale - Blue sclera : pulmonary TB - Fundus : Choroidal nodules in miliary TB, papilledema in hypercapnea - Phlyctenular conjunctivitis in TB - SubConjunctival hemorrhage in chronic cough - Horner's syndrome : Pancoast tumor, mediastinal syndrome 2) Nose & Cheeks: - Working Ala nasi : severe dyspnea in pneumonia - Plethoric face : polythycemia in COPD 3) Mouth & Throat: - Mouth odour : fetid odour in SLS - Tongue : central cyanosis	Enlarged cervical LN especially scalene LN : TB, sarcoidosis, bronchial carcinoma. Congested neck veins : Pulsating : - RVF (cor pulmonale) ↑ Intrathoracic pressure in : pneumothorax, emphysema Non pulsating : - Mediastinal syndrome (SVC obstruction) - Severe RVF (cor pulmonale)
❖ Upper limb	❖ LL
Tremors : A. Fine tremors : B stimulant used in treatment of bronchial asthma B. Flapping tremors (asterixis) : respiratory failure Clubbing : a) Hypoxic: - COPD; clubbing usually mild – marked in associated bronchiectasis - Interstitial pulmonary fibrosis b) Toxic: - Suppurative lung syndromes - Fibroid type of pulmonary TB - Bronchogenic carcinoma - Pleural methothelioma c) Unilateral : Pancoast tumor	Chest Causes of LL oedema: RVF: due to corpulmonale Hypoalbuminemia: - Lost purulent sputum in SLS - Frequent aspiration of empyema - Nutritional : anorexia (↓ protein intake) Nephrotic syndrome : renal amyloidosis in long standing SLS Acid-base disturbance: Renal regulation in COPD : ↑PCO2 leads compensatory ↑ NaHCO3 reabsorption from kidney, with H2O retention Cortisone therapy for BA

IV. Other systems except that of local exam :

❖ Cardiac examination	❖ Abdominal examination	❖ Neurologic examination
A. As a cause : - Pulmonary congestion - Pleural effusion - Left side heart failure, fine bilateral basal crepitation B. As a result : cor-pulmonale C. As an association : Kartagener syndrome	A. Hepatomegaly : may occur due to - Cor pulmonale : Bilharzial, hypoxic - Amebic liver abscess - Amyloidosis : chronic toxemia - Secondaries from bronchial carcinoma - Fatty changes : chronic toxemia - Associated liver disease : viral hepatitis - Ptosed liver (downward displacement) in emphysema (palpable liver) B. Ascites : corpulmonale "Bilharzial, hypoxic" C. Splenomegaly: - Corpulmonale : Bilharzial - Hypoxic (with cardiac cirrhosis) - Miliary TB, Sarcoidosis & Amyloidosis	- Neuropathy or myopathy as paramalignant syndrome in bronchogenic carcinoma - Pott's disease - Meningeal irritation (T)

Local Examination Of Chest Case

Rules :

1. Good light
2. Exposure the patient's chest from umbilicus upwards.
3. Position of the patient supine with the head of the table slightly flexed
4. Always examine from the patient's right side
5. Make sure that the patient is comfortable in this position

Examination:

I. Inspection	II. Palpation	III. Percussion	IV. Auscultation
○ Area ⇔ Area	○ Area ⇔ Area	○ Space ⇔ Space	• أثناء الفحص يتم الفحص بمقارنة كل area بالأخرى أو كل space بآخر : مسموح بالاتنين ○ Area ⇔ Area or ○ Space ⇔ Space

قوانين الفحص :

- إبدأ من normal healthy side
- لو المرض bilateral إبدأ بأي ناحية , بس بكل فحص إبدأ بالناحية اللي اخترتها
- مقارنة كل Aspect بالأخرى :
 - العيان نايم وأيده جنب منه front ⇔ front
 - العيان نايم وأيده فوق راسه lateral ⇔ lateral
 - عند فحص ال back ⇔ back :
 - العيان جالس وأيده بجانبه : supra-scapular area
 - العيان جالس ومربع أيده أو حاضن نفسه inter-scapular & infra-scapular

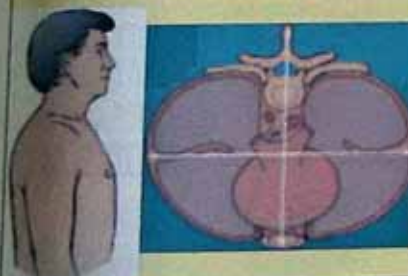
I. Inspection

1) Shape & Symmetry:



- أوقف من عند حرف السرير وبص tangential وقول للعيان وقف نفسك وشوف تضاريس منطقة الصدر
- وقارن كل area بالأخرى supra-mammary area, mammary area & infra-mammary area
- هل هي متساوية symmetrical ولا فيه حثة نازلة وحته طالعة asymmetrical ?

A. Normal symmetrical chest



- Shape : elliptical
- Diameter: Antro-posterior < transverse diameter (5 : 7)
- Subcostal angle: $\leq 90^\circ$
- Ribs : oblique
- Intercostal space: narrow normal
- Spine , vertebral column : normal curvature

I. Symmetrical deformities



1) Barrel (emphysematous) chest

- Shape : barrel & symmetrical bilateral hyperinflated chest
- Diameter : Antro-posterior diameter $\geq$ transverse diameter
- Subcostal angle: wide $> 90^\circ$
- Ribs : horizontal
- Intercostal spaces: wide
- Shoulders : raised
- Sternum : prominent
- Spine : Kyphosis

B. Abnormally :

II. Asymmetrical deformities



2) Unilateral retraction

3) Unilateral bulge

A. Differentiated by chest expansion (diseased part moves less)

B. Causes :

- Fibrosis
- Collapse
- Pleura : Pleural effusion, pneumothorax, empyema necessitans
- Lungs : unilateral emphysema
- Cardiac : precordial bulge (RV ++, pericardial effusion)
- Chest wall : Subcutaneous emphysema

Other abnormalities of the shape :

Symmetrical deformities			Asymmetrical deformities
❖ Flat chest "alar chest"	❖ Pigeon's chest "pectus carinatum"	❖ Funnel chest "pectus excavatum"	❖ Thoracic kyphoscoliosis
			
○ Cause : fibrosis as TB ○ Winged scapula ○ Decreased AP diameter ○ Ribs : More oblique	○ Bone softening diseases: Ricketts, Marfan syndrome ○ Prominent sternum with forward protrusion, ○ $\uparrow$ A-P > transverse diameter	○ Congenital ○ May be acquired in shoemakers ○ Inward depression of lower part (or the whole) sternum ○ Effect: compress RV with ischemia of lung	○ Common causes : osteoporosis, osteoarthritis & TB ○ Curved spine & chest shows the corresponding deformities ○ Kyphosis : abnormal curvature of spine ○ Scoliosis : abnormal lateral curvature of spine ○ Effect : cor pulmonale

2) Chest expansion:



وقف من عند حرف السرير وبص tangential وقول للعيان خذ نفس جامد من بقاء وخرجه
تلف الصدر **ببتحرك** كويس ولا لا ؟

ولا : هل الحركة قلت على الناحيتين ولا ناحية واحدة ؟

على الناحيتين : bilateral limitation

على ناحية واحدة يبقى : unilateral limitation : الناحية المصابة هي التي بتتحرك أقل مع النفس

❖ Normally	❖ Abnormally : limitation of movement detect the diseased	
▪ Chest moves freely with respiration ▪ Accurate done by tape test; see later	▪ Bilateral limitation	▪ Unilateral limitation
	▪ In bilateral chest disease e.g. Emphysema, bronchiectasis	▪ On the affected side e.g. Fibrosis, collapse.

3) Chest wall abnormality; examine the front, sides & BACK of the chest

1. Skin lesions: e.g. pigmentation, Campbell de Morgan spots, spider nevi, ulcers, ... etc.
2. Scars :



Chest Tube



Aspiration of pleural effusion

- Intercostal chest tubes : over axillary areas
 - Aspiration of pleural effusion : over the back in infra-scapular region
3. Dilated veins filling from above downwards in SCV obstruction & mediastinal syndrome
 4. Ribs & intercostal space:

❖ Normally :	❖ Abnormally:	
▪ Normally oblique	▪ In emphysema horizontal	▪ In fibrosis more oblique

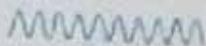
4) Respiratory Movement:

1. Type of respiration :

❖ Normally		❖ Abnormally	
- Inspiration is affected by contraction of intercostal muscles & diaphragm - Expiration is a passive process, dependent on elastic recoil of lungs - In females : respiration is predominantly thoracic (Thoraco-abdominal) - In males : respiration is predominantly abdominal (Abdomino-thoracic)		Abdominal respiration	Thoracic respiration
		❖ Causes	
	- Intercostal muscle paralysis (poliomyelitis) - COPD, Pleurisy, pleural effusion - Ankylosing spondylitis	- Phrenic nerve paralysis "diaphragmatic paralysis" - Abdominal distention (ascites, pregnancy) - Peritonitis	

2. Respiratory rate :

- Normal rate : 12 – 20 /min



(pulse : respiration ratio = 4 : 1)



عدده من صدر أو بطن العيان في دقيقة كاملة واعمل نفسك بتعد ال Pulse

Abnormality in rate :

A. Bradypnea: below 12 ↑ ICT, inhibition of RC (morphine, alcohol)	B. Tachypnea: above 20 All causes of acute dyspnea & fever
--	--

3. Rhythm :

- Normal rhythm : regular and quiet with expiratory phase lasts about 1/3 as long as inspiratory phase

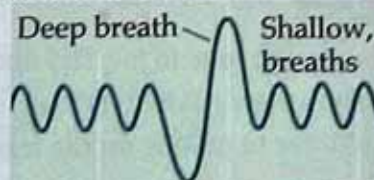
Abnormality in Rhythm:

A. Cheyne – stokes breathing	B. Biot's (ataxic) breathing
❖ Definition:	
A period of apnea for 1/2 min., followed by gradual hyperpnea then gradual hypopnea till apnea occur & so on	Unpredictable irregularity breaths may be shallow or deep & stop for short periods
❖ Causes:	
↓ Sensitivity of RC to Co₂ as in : - Elderly (during sleep) - Inhibited RC : cerebral atherosclerosis, respiratory Failure, narcotics, ↑ ICT	- Meningeal irritation e.g. meningitis - Subarachnoid hemorrhage

4. Depth :

- Normal depth : average i.e. Full expansion of the chest wall with full relaxation

Abnormality in depth:



A. Hyperpnea (rapid deep)	B. Hypopnea (shallow)
- Acidosis "Kussmaul's respiration" in DKA & Chronic Renal Failure - Psychogenic (sighing respiration) - Exercise, emotional stress	- Rapid shallow : left side HF, pneumonia - Slow shallow: Inhibited RC by drugs e.g. barbiturates

5) Pulsations : see cardiology

- A. Apical pulsations, Pulmonary pulsations for pulmonary hypertension, Left parasternal pulsations
- B. Epigastric pulsations; in cor pulmonale, RVE is diagnosed by epigastric pulsation (source from above).

6) Position of mediastinum:

A. Apex

Value of apex in chest cases:

- A. Chest causes of Shifted apex:
 - Pulling of apex : fibrosis & collapse
 - Pushing: pleural effusion, pneumothorax
 - Upward : diaphragmatic paralysis
- B. Chest causes of Absent apex :
 - Obesity, behind a rib
 - Emphysema, left sided pneumothorax or Pleural effusion
- C. In corpulmonale : RVE (shifted out + diffuse)
- D. Congenital dextrocardia :
 - In Kartagener syndrome "immotile cilia syndrome": absent frontal sinuses or frontal sinusitis, congenital bronchiectasis, decrease ciliary movement: infertility , dextrocardia



B. Trachea

- A. Normally trachea is central with both tendon of sternomastoid is symmetrical in shape & position
- B. Abnormally: shifted;
 - Sternomastoid (Trail's) sign: in THIN patients: unilateral bulge of sternomastoid tendon on side of tracheal shift due to displacement of trachea behind the tendon.

يفضل والمريض جالس ، القي نظريتين على tendons ال مرة من الأمام ومرة من الجنب

7) Signs of respiratory distress:

❖ Comment on SACT

I. Sign of respiratory distress:

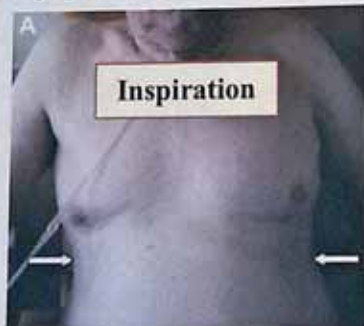
1. Littin's Sign :



❗ Littin's Sign ≠ Litten's sign ال فرق ما بين ال

- Normally there is Inspiratory indrawing of lower intercostal space on chest sides due to diaphragmatic descent
- It's exaggerated in obstructive lung diseases (marked inspiratory indrawing & expiratory fullness of ICS)
- It's absent in obesity, pleural effusion, diaphragmatic paralysis

2. Hoover's Sign:



❗ Remember : 2 Hoover sign :

1. Chest : Inspiratory indrawing of costal margin in COPD due to low flat diaphragm
2. Neurology: in hemiplegia to differentiate between organic and hysterical causes (See later)

- Inspiratory indrawing of costal margin in COPD; due to low flat diaphragm (reverse of the normal)
- 3. Campbell's Sign or Tracheal Tug with inspiration or false tracheal tug:
 - Descent of cricoid cartilage with inspiration due to pulling on the trachea during inspiration by strong diaphragmatic contraction in COPD
 - Patient head is extended, the tip of index is placed below cricoid cartilage with slight pressure upwards - sudden jerky downwards movement of trachea (tugged) sufficiently to squeeze the finger can be felt with inspiration
- 4. Crico-Sternal distance = tracheal length:
 - Technique to measure this space, insert two or three fingers between the cricoid cartilage and the suprasternal notch
 - Normally: the trachea can be palpated from the cricoid to the suprasternal notch (crico-sternal distance is 3-4 fingerbreadth.
 - Abnormally: If less than 3 finger breadths; this indicates hyperinflation of the lung (COPD).

II. Manifestations of respiratory failure: Types II, ($O_2 < 60$ mmHg, $Co_2 > 50$ mmHg)

1. Working **Ala nasi**
2. Abnormal movement of **Accessory muscles**:

☐ Normally :

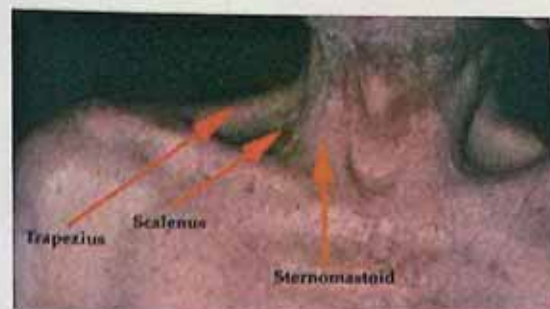
- No over action of accessory muscles of respiration
- Muscle of inspiration : diaphragm & intercostal muscles
- No muscles for expiration as it is passive process

☐ Abnormally:

A. Abnormal inspiratory movements:

- 1) Contraction of inspiratory accessory muscles :
 - Trapezii, Scalenus muscles, Sternomastoids
- 2) Thoracic up lift :
elevation of thoracic cage due to sternomastoid contraction
- 3) Exaggerated indrawing of:
 - Supraclavicular fossae
 - Suprasternal
 - Intercostal spaces

Respiratory failure is a laboratory diagnosis (ABG)



B. Abnormal expiratory movements:

1. Contraction expiratory accessory muscles:
 - Abdominal muscles
 - Latissimus dorsi
2. Tri-pod position: Patient sit upright & grasp a bed table or back of a chair
 - To fix shoulder girdle, so that latissimus dorsi can be used for approximating ribs
 - Augmenting expiratory efforts.
3. Pursing lips :
 - To keep intrabronchial pressure high above that of surrounding alveoli
 - And so preventing collapse of bronchial wall during expiration
3. Co_2 narcosis {(hypercapnic encephalopathy): hypersomnia & drowsiness},
4. Central Cyanosis
5. Clubbing
6. Flapping Tremors (asterixis)
7. Tachypnea
8. Tachycardia



II. Palpation

1) Position of mediastinum:

A. **Apex:** see cardiology

B. **Trachea:**

❖ **Technique :**

- Ask the patient to sit & centralize his head using your left hand on his vault
- Try to insinuate your index finger with its PALMER aspect between trachea & sternomastoid tendon on sides
- **Normally:** central : same distance on both sides
- **Abnormally:** shifted : unequal distance being narrow at the side of tracheal deviation

❖ **Causes of mediastinal shift:**



A. Shift of apex alone	B. Shift of trachea alone	C. Shift of trachea & apex :
▪ Cardiomegaly ▪ Dextrocardia ▪ Moderate pleural effusion	▪ Superior mediastinal tumor ▪ Retrosternal goiter	▪ Pulled to same side : ○ Fibrosis, collapse ▪ Pushed to opposite side ○ Effusion, pneumothorax

2) Tenderness:

A. Palpate gently over the chest wall while inspecting the patient's face.

B. Causes of tenderness over chest wall :

- Over ribs : fracture, inflammation, tumors, Tietz syndrome (costo-chondritis)
- Over intercostal spaces : myalgia, neuralgia (Herpes Zoster; unilateral & dermatomal), pleurisy, empyema
- Over sternum : leukemia



3) Chest expansion:

I. Accurate method: Tape test :

Put the tape around the chest wall, at or below fourth intercostal space (i.e. at the level of the nipple), with zero of the tape at anterior midline

Measure chest circumference at full expiration & full inspiration,:

- Normally: the difference should be 3 – 5 cm
- Abnormally: ≤ 2.5 cm there's generalized restriction of chest mobility as COPD



II. Other method:

A. Expansion of lung zones :

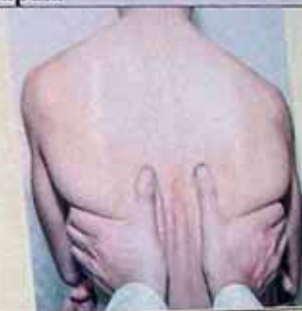
1. Upper lung zone (including lung apices), above the second rib in supra-mammary region

2. Middle lung zone, from the second rib to the fourth rib in mammary region

3. Lower lung zone, below fourth rib in infra-mammary region

B. Expansion of the back :

❖ Infra-scapular area at level of T10 i.e. just below the angle of scapula



العيان نايم supine

ضم صوابك على بعض ما عدا ال thumb واحط ايدي الاتنين على ال chest كل واحد في ناحية بحيث تكون أطراف الأصابع على ال clavicle وال 2 thumbs على نفس المستوى وباصيين للمسقف

اطلب من العيان ان ياخذ نفس من بقة ولاحظ حركة اليد لفوق وتحت antro-posterior

العيان نايم supine

ضم صوابك على بعض وابعدهم عن ال thumb وخليها تلف حوالين صدر العيان وحط كل ايد على جنب العيان بحيث يكون ال 2 thumbs في المنتصف عند مستوى ال mammary region or infra-mammary region

قرب ايديك الاتنين من بعض بحيث تاخذ thin fold of skin بين ال 2 thumbs اطلب من العيان ان ياخذ نفس من بقة ولاحظ ابتعاد ال 2 thumbs عن بعض لثناء ال inspiration المفروض يبقى أكبر من 5 سم

تتعمل في ال infrascapular بـ
وبنفس طريقة ال fold of skin السابقة
ولكن العيان قاعد ومربع ايديه

❖ Unilateral limitation
as seen associated with e.g. Fibrosis, collapse, pleural effusion, pneumothorax & pneumonia

Causes of limited chest expansion :

Unilateral limitation

4) Subcostal angle:

- Technique : thumb test
- Normally : acute to right angle (70° – 90°)
- Abnormally: Obtuse angle: emphysema, upper abdominal swellings & ascites



5) Pulsations: see cardiology

- Confirm pulsations (apex, pulmonary, left parasternal pulsation, epigastric) : see cardiology.
- Importance of that pulsation in chest case: this may diagnose cor-pulmonale

• Cor- pulmonale : Parenchymal or vascular lung disease → P.HTN → RVE → ± RV failure

✦ **Manifestation of cor-pulmonale :**

- ☐ Manifestation of **primary chest disease** : Symptoms + Signs (see before)
- ☐ Manifestation of **pulmonary hypertension** : Symptoms + Signs (see cardiology)
- ☐ Manifestation of right ventricular **enlargement** and or right ventricular **failure** :

❖ RVE	❖ RV failure
▪ Left parasternal pulsation : maybe <i>not felt</i> as mostly the patient had emphysema ▪ Apex pulsation : weak or <i>absent</i> as mostly the patient had emphysema, but if detected it will be diffuse and shifted outwards ▪ Epigastric pulsation : may be detected due to RVE or liver congestion as a result of cor-pulmonale	▪ Congested pulsating neck veins <i>with expiratory filling</i> ▪ LL edema ▪ Enlarged tender pulsating liver ▪ Ascites ▪ Tricuspid gallop & functional tricuspid incompetence murmur: it's specific for RVF

6) Tactile vocal fremitus (TVF):

❖ Definition:

- Palpable vibration of vocal cords transmitted through respiratory passage to be felt on chest wall.
- It is done by applying hand on chest wall and ask patient to repeat "99" or in Arabic "44"

❖ Normally :

- TVF is equally on in each two corresponding areas of the chest **except**:
Right 2nd space in parasternal line (Right upper lobe); TVF is increased **because** :
 - Trachea is in contact with Right lung apex
 - Right apical bronchus is more superficial (superior)

❖ Abnormally :

❖ Causes of increased TVF = causes of bronchial breathing = causes of ↑ vocal resonance	❖ Causes of decreased TVF
▪ <u>Consolidation</u> (pneumonia) ▪ <u>Cavity</u> if : big, superficial & surrounded by consolidation ▪ <u>Collapse</u> or fibrosis if associated with patent bronchus ▪ Upper level or pleural effusion	▪ All other chest diseases: Causes of ↓ TVF = causes of ↓ intensity of breath sounds = causes of ↓ vocal resonance (see later)

❖ Technique : see below

❖ Comment on SACT:

7) Palpable adventitious sounds:

1. Site: = causes of Palpable Ronchi:

- Generalized e.g. bronchitis or bronchial asthma
- Localized e.g. partial obstruction by tumor or foreign body or secretions

2. Which Adventitious sound :

- Palpable ronchi in bronchitis (DD with thrill by stopping breathing)
- Palpable rub in pleurisy
- Palpable crepitations

3. Relation to Cough:

- Persist or disappear due to secretions

4. Timing:

- Inspiratory or expiratory or both

❖ Technique : see below

6) Tactile vocal fremitus (TVF)

- ❖ **Technique:** By the same hand, comparative; area by area in each two corresponding symmetrically areas (Right to Left) by placing the hand ball of the palm while tips directed laterally.
- ❖ Compare intensity of vibrations while the patient is repeating "99" or in Arabic "44"
- ❖ Some schools may feel the TVF by the ulnar border of hand

اطلب من العيان انه يقول اربعة اربعة في كل منطقة
اطلب من العيان انه ياخذ نفس من بقة في كل منطقة SACT
Ask the patient to take deep respiration and then comment on SACT

1. Anteriorly: BOTH SIDES : العيان نايم وايدة جنبه

A. Supra-mammary = upper 1/3 (oblique (ايد الدكتور تبقي



B. Mammary = middle 1/3 (horizontal (ايد الدكتور تبقي



C. Infra-mammary = lower 1/3 (horizontal (ايد الدكتور تبقي



2. Laterally: BOTH SIDES

العيان نايم وايدة فوق راسه

A. Upper axillary (upper 1/2) (horizontal & fingers towards the bed)



B. Lower axillary (lower 1/2) (horizontal & fingers towards the bed)

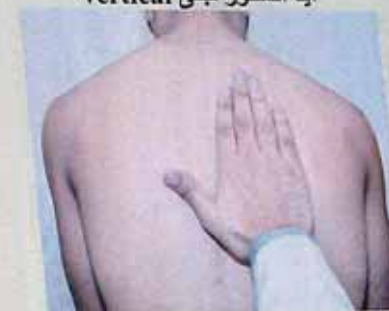


3. Posteriorly : BOTH SIDES العيان جالس

A. Supra-scapular (above spine) Oblique (ايد الدكتور تبقي



B. Inter-scapular (between scapulae) vertical (ايد الدكتور تبقي

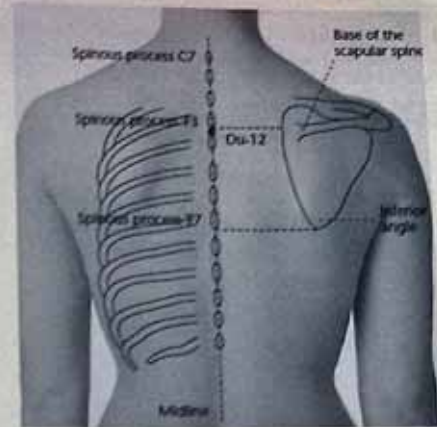


C. Infra-scapular (below scapula) horizontal (ايد الدكتور تبقي



III. Chest Percussion

- I. Hepatic dullness (see cardiology)
- II. Heart percussion (see cardiology)
- III. Chest wall:
 - A. **Lung proper** : Light percussion, except back by heavy percussion
 - o Lung (front, side, back)
 - B. **Special areas by light percussion** :
 1. Bare area of the heart: see cardiology
 2. Kronig's Isthmus
 3. Traub's area



❖ Revision of the anatomy :

→ The scapula covers from 2nd to 7th rib with the arm at the sides and the inferior angle of scapula is usually at the 7th intercostal space.

❖ Grades of percussion note:

A. Resonance			B. Dullness		
1. Normal resonance	2. Hyperresonance	3. Tympanitic resonance	1. Slight dullness (Impaired note)	2. Dullness	3. Stony dullness
❖ Causes					
▪ Normally: - o Lung & free abdomen	▪ Emphysema : encroach the normal dullness of bare area and or upper hepatic border due to hyperinflation of the chest.	▪ Air: - o Empty stomach - o Pneumothorax - o Lung cyst - o Emphysematous bullous	▪ Normally : scapulae ▪ Abnormally: - o Minor - o Consolidation - o Collapse - o Fibrosis	▪ Normally : - o Heart, liver ▪ Abnormally: - o Massive: - o Consolidation - o Collapse - o Fibrosis	Pleural effusion

❖ Areas of percussion :

• Anterior



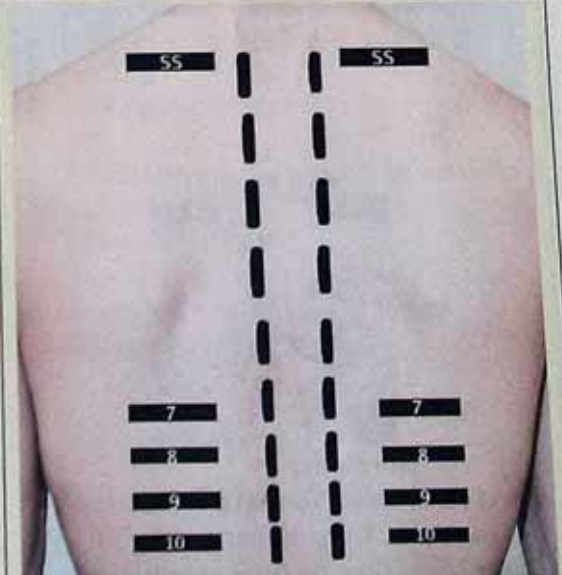
- K : Kronig's isthmus
- C : Clavicle
- I : Infra- clavicular
- Numbers : Spaces

• Lateral



- Numbers : Spaces

• Posterior



- SS : Supra-Scapular
- Vertical Lines: Inter-scapular
- Horizontal lines : Infra-Scapular

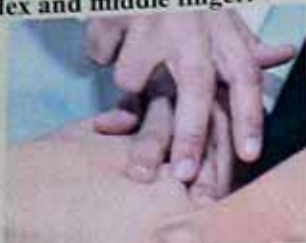
1) Lung areas, on both sides simultaneously, comparative: area by area:

1. Anteriorly mainly in MCL by light percussion:

العيان نايم وايدة جنبه

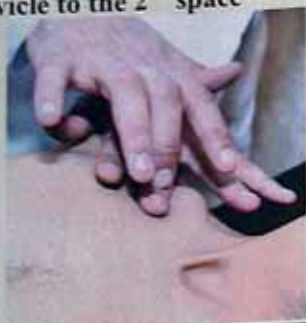
A. Clavicle;

- Direct percussion over medial third of the clavicle, stretch the skin by index and middle finger.



شد الجلد من على ال clavicle وضع صباعين ال middle finger & index على شكل حرف تمانية وخط بايديك اليمين (ده عشان صباعك ميترحلقتش)

B. Infra-clavicular region: from the clavicle to the 2nd space



C. Space by space:

Both sides from 2nd → 6th at MCL except left 4th & 5th spaces at AAL

2 ⇔ 2
3 ⇔ 3
4 ⇔ 4
5 ⇔ 5

End of lung



Normally:

- At right side: Normal dullness at 6th space (MCL) by upper border of the liver. then do tidal percussion to make sure
- At left side: at 6th space (MCL) ↑ resonance: Traub's area "tympanitic resonance"

Abnormally:

- Higher level of lung resonance: pleural or pulmonary disease.
- Lower level of lung resonance: below 5th space in MCL, in emphysema, pneumothorax

2. Laterally mainly in MAL by light percussion

العيان نايم وايدة فوق راسه

4th space → 7th space

4 ⇔ 4
5 ⇔ 5
6 ⇔ 6
7 ⇔ 7

End of lung



قارن كل space ⇔ space بداية من الرابع لانه اول space تقدر توصله والافضل انك تعد من الامام وتمشي معاه lateral العيان

Normally:

- At right side: Normal dullness at Right 8th ICS (MAL) by upper border of the liver. then do tidal percussion to make sure
- At left side: at Left 8th ICS (MAL) ↑ resonance: Traub's area "tympanitic resonance"

3. Posteriorly by heavy percussion

الموصل تغيريين:

العيان جالس والدكتور ايدة تخبط جامد

A. Supra-scapular (Trapezius): above spine of scapula



العيان قاعد وايدة جنبه

horizontal تخبط جامد وتبقى

B. Inter-scapular: in paravertebral line From 2nd to 10th space



العيان قاعد ومربع ايدة او حاضن نفسه

vertical تخبط جامد وتبقى

C. Infra-scapular: in scapular line From 7th (scapular angle) → 10th space



العيان قاعد ومربع ايدة او حاضن نفسه

horizontal تخبط جامد وتبقى

Normally:

- At right side : Normal dullness Right 10th ICS in scapular line upper border of the liver. then tidal percussion to make sure
- At left side : Normal dullness 10th ICS in scapular line by upper border of spleen

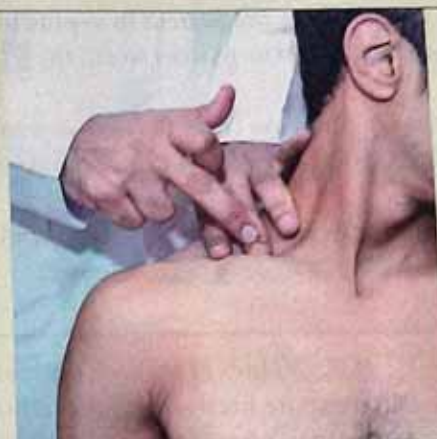
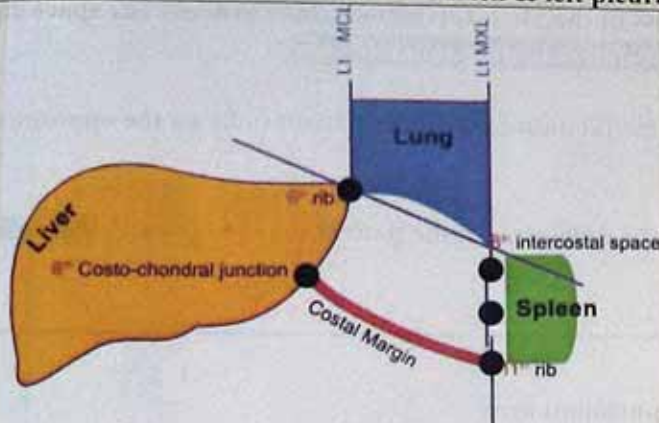
2) Traub's area

3) Kronig's isthmus

❖ Definition :

- An area of tympanitic resonance overlying air bubbles in fundus of stomach
- Normally: tympanitic resonance
- Contents of Traub's area: fundus of stomach & left pleura

- A band of resonance overlying lung apex
- Normally: resonance



Boundaries & surface anatomy:

1. **Right border** : left border of liver :
 - Left 6th rib MCL → left 8th costal cartilage

2. **Upper border** : lower border of Left lung:
 - Left 6th rib MCL → left 8th rib MAL

3. **Lower border** : left costal margin:
 - left 8th costal cartilage → left 11th rib MAL

4. **Left border** : anterior border of spleen:
 - Left 9th rib MAL → left 11th rib MAL

- 1) **Medially**: a line connecting the spine of C7 vertebra to sternoclavicular joint
- 2) **Laterally**: a line connecting midpoint of spine of scapula to junction of medial 2/3 with lateral 1/3 of clavicle
- 3) **Anteriorly**: medial 2/3 of clavicle
- 4) **Posterior**: trapezius muscle

borders Traub's area متجها لل borders

المريض جالس ويثني راسه والدكتور من الخلف وخبط من جوه لبره

- Technique: by light percussion from center to borders.

- Technique: by light percussion from dullness to resonance

Abnormally

Causes of Dullness of Traub's area:

Causes of Increased size of resonance of Traub's area:

Stomach

- 1) Full stomach or gastric tumors

A. Dilated stomach

From above

- 2) Lt basal pleural disease (effusion, thickening)
- 3) Pericardial effusion

B. Lt. Basal lung collapse or pneumonectomy

From below

- 4) Elevated diaphragm (ascites, tumor & pregnancy)

C. Pneumoperitoneum

From right

- 5) Hepatomegaly (enlarged left lobe)

D. Shrunken liver

From left

- 6) Splenomegaly

E. Splenectomy

Congenital

- 7) Situs inversus totalis

F. Congenital eventration of diaphragm

Abnormally : dullness in Kronig's isthmus → apical lesion, causes include:

- a) Apical lung lesions :
 - TB
 - Friedlander's Pneumonia (Klebsiella pneumonia)
 - Fibrosis - collapse
- b) Apical lung tumor: Pancoast tumor
- c) Apical pleural lesions : Pleural thickening

❖ NB:

- Physiological causes of dullness of Traub's area: full stomach & pregnancy.

❖ Special test: Shifting dullness : to diagnose hydropneumothorax by percussion:

- Value : differentiate between **pleural effusion** & hydropneumothorax
 - Technique : we percuss in 3 plains while changing patient position:
- 1) 1st plane : while the patient in supine position , percuss in the **MCL** till dullness, then proceed one space above (resonant), ask the patient to **sit**, the **resonance will be dull in hydropneumothorax**
 - 2) 2nd plane : percuss **from the sternum to the axillary line** till dullness, ask the patient to lie on the **opposite side**, the **dullness will be resonant in hydropneumothorax**
 - 3) 3rd plane : percussion on the **back (scapular line)** till the dullness, ask the patient to **lean forward**, the **dullness will be resonant in hydropneumothorax**

🐼 N.B.: Values of shifting dullness:

- 1) Differentiate between pleural effusion & hydropneumothorax
- 2) Differentiate between pericardial effusion & pulmonary dilatation as a cause of dullness in pulmonary area
- 3) Ascites

IV. Auscultation:

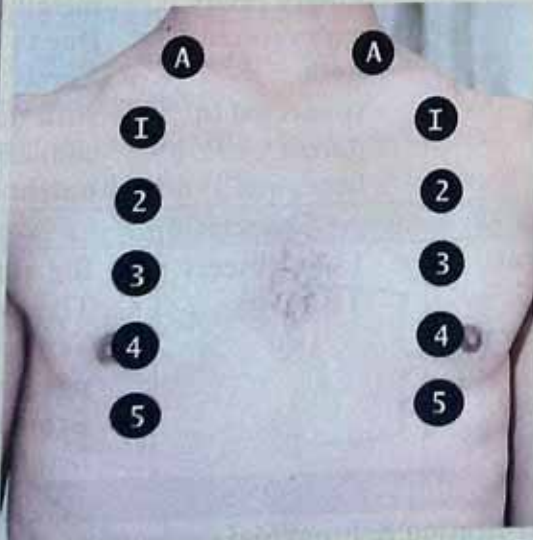
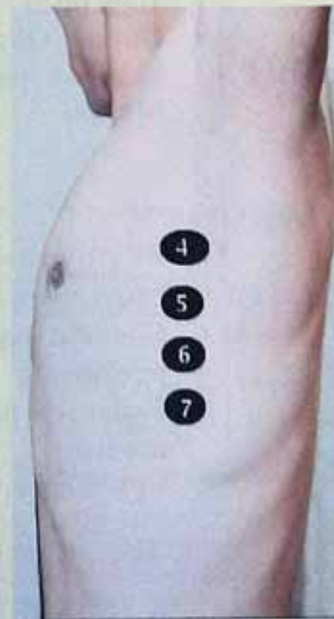
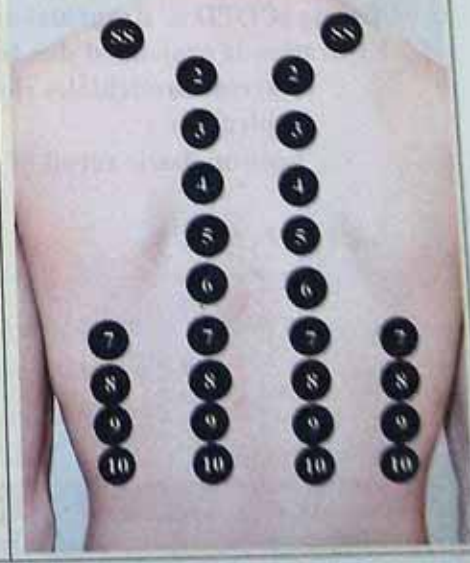
A. Auscultation for:

- 1) Breath sounds: Vesicular, Bronchial & Broncho-vesicular.
- 2) Conducted sounds: Vocal resonance (Bronchophony, Whispering pectoriloquy, Aagophony)
- 3) Adventitious sounds: Pleural rub, Crepitations & Wheezes
- 4) Special test: Post- tussive suction, Succession splash & Coin test.

B. Parts of stethoscope used for the sounds :

- 👉 We can use **either** diaphragm **or** cone (bell) but the **CONE (BELL) IS PREFERRED** due to:
- Most of the sounds reaching the chest wall from the bronchi and lungs are of low pitch.
 - To avoid falsely produced sounds as pleural rub or crepitations resulting from stretching of the skin and when using the diaphragm of stethoscope during deep breathing

C. The maneuver of auscultation: comparative on both sides either area by area or space by space:

	1. Anterior aspect العيان نايم وايدة جنبه	2. Lateral aspect العيان نايم وايدة فوق راسه	3. Posterior aspect العيان جالس
A. By areas	❖ In midclavicular line: ○ Supramammary areas  ○ Mammary areas  ○ Inframammary areas 	❖ In mid axillary line: ○ Upper axillary areas  ○ Lower axillary areas 	○ Supra-scapular above spine of scapula  ○ Interscapular areas in PVL  ○ Infrascapular areas in SL 
B. By spaces	▪ Apex of the lung ▪ Infra clavicular ▪ From 2nd to the 5th space in MCL 	▪ From 4th to 7th space 	▪ Supra-scapular ▪ Interscapular from 2nd to 10th space in paravertebral line ▪ Infrascapular from 7th to 10th space in scapular line 

1) Breath sounds:

A. Intensity = loudness of breath sounds:

- 1) Normal intensity : louder in lower posterior lung field
- 2) Increased intensity = loud intensity of breath sound : thin chest & children (puerile breathing)
- 3) Decreased intensity = diminished intensity of breath sound = Diminished air entry; due to :
❖ Causes of diminished intensity of breath sounds = decreased vocal resonance = decreased TVF :
 - a) Reduced air flow : Severe late cases of COPD, Bronchial asthma, Collapse & fibrosis
 - b) Reduced conduction: Obesity, Pleural effusion & Pneumothorax

B. Types of Breath sounds:

i. Vesicular breathing "alveolar breathing"

- Flow of air in and out the normal lung alveoli

- Rustling حفيف الشجر = soft خفيف = breezy نسيم (lower pitch)



- Inspiration is longer than expiration (3:1)
- No gap between inspiration & expiration

❖ Mechanism:

- Alveoli out of work + Patent bronchi

❖ Characters:

- Hollow = blowing (higher pitch)
- Expiration is $\geq$ Inspiration
- There is gap between inspiration & expiration



❖ Heard over :

- ☞ Normally:
 - Heard over the lungs & Best heard over axilla & infrascapular regions
- ☞ Abnormally:
 - Change in character: Expiration is prolonged → Vesicular breathing with prolonged expiration

☞ Characters of Vesicular with prolonged expiration :

- Character : Rustling , No gap & expiration is prolonged & usually wheezy
- Causes: Occur in obstructive airway disease (COPD & Bronchial asthma)
- Expiration is prolonged due to:
 - Narrow bronchioles slows the expiration
 - Loss of elastic recoil of the lung.

- ☞ Normally heard over trachea & large airway
- ☞ Abnormally heard over : = causes of $\uparrow$ TVF = causes of $\uparrow$ vocal resonance

- Consolidation
- Cavity if: big, superficial & surrounded by consolidation
- Collapse, fibrosis if associated with patent bronchus
- Upper border of pleural effusion (rare)

- Confirmed by: $\uparrow$ vocal resonance (especially whispering pectoriloquy)

❖ Classification of bronchial breathing according to pitch in 3 types :

1) Tubular

2) Cavernous:

3) Amphoric

❖ Definition:

- | | | |
|--|--|--|
| <ul style="list-style-type: none"> ▪ High pitch ▪ Due to : Obstructed alveoli & patent bronchi | <ul style="list-style-type: none"> ▪ Low pitch ▪ Due to <ul style="list-style-type: none"> ○ Empty cavity with relaxed walls connected to patent bronchus | <ul style="list-style-type: none"> ▪ Low pitch with superadded metallic tone ▪ Due to : <ul style="list-style-type: none"> ○ Empty cavity with tense walls connected to patent bronchus |
|--|--|--|

❖ Causes:

- | | | |
|--|---|--|
| <ul style="list-style-type: none"> ▪ Consolidation as pneumonia ▪ Collapse + patent bronchus ▪ Upper border of Pleural effusion | <ul style="list-style-type: none"> ▪ Lung abscess ▪ TB cavity | <ul style="list-style-type: none"> ▪ Big abscess ▪ TB cavity ▪ Tension pneumothorax ▪ Bronchopleural fistula |
|--|---|--|

iii. Broncho-vesicular breathing:

- ☞ Combination of vesicular & bronchial types e.g. vesicular inspiration & bronchial expiration.
- ☞ Normally in right lung
- ☞ Abnormally in patchy consolidation



2) Vocal resonance

- التنفس الطبيعي يتألف من الـ inspiration أطول من الـ expiration بنسبة 3 : 1
- والتنفس الطبيعي والمرضي يتألف من الـ bronchial breathing أن الـ expiration أطول من (أرسلوا) الـ inspiration الذي ينقل عليه
- والتنفس المرضي يتألف من الـ vesicular breathing أن الـ expiration أطول من الـ inspiration الذي ينقل عليه
- vesicular breathing with prolonged expiration
- فدلوقتي الدكتور لو يسمع بالسماعة ولاقي ان الـ expiration أطول من الـ inspiration فهو اداها احتماليين يا اما هو
- bronchial breathing او انه vesicular breathing with prolonged expiration
- وهنا نتيجي اهمية الـ vocal resonance : التي هي اختبارات معينة بتعملها مع العيان وتثبت ان اللي خلى الـ expiration اكبر من الـ inspiration هو الـ bronchial breathing

- **Definition:** it's vibration of vocal cords transmitted through airway and chest wall to be auscultated
- **Value:** Conducted voice sound, confirm bronchial breathing
- **Technique:** Tested while patient says 44 in Arabic, one one or nine nine in English, compare equivalent areas of chest

❖ Types of vocal resonance

A. Bronchophony	B. Whispering pectoriloquy	C. Aagophony
▪ العيان هيقل أربعة أربعة بصوت عادي ▪ الدكتور هيسمع الصوت أعلى في مكان الـ bronchial breathing	▪ العيان هيقل أربعة أربعة بصوت منخفض او بهمس ▪ الدكتور هيسمع الصوت أعلى في مكان الـ bronchial breathing	▪ العيان هيقل أربعة أربعة بصوت عادي ▪ الدكتور هيسمع الصوت مخفف في مكان الـ bronchial breathing
○ Normally spoken words are heard louder & clearer	○ Whispered sound are heard border & clearer	○ Normally spoken words are heard louder with nasal tone
▪ Causes of increased vocal resonance : same causes of $\uparrow$ TVF = same causes of bronchial breathing ▪ Causes of decreased vocal resonance : same causes of $\downarrow$ TVF = same causes of diminished intensity of breath sounds		

❖ N.B.: D'espine sign:



- **Normally :** if you auscultate below the body of T4 (tip of T2 spine), you will hear vesicular breathing
- **Abnormally :** if you heard Bronchial breathing confirmed by $\uparrow$ of vocal resonance
- **Significance:** Indicates enlarged mediastinal (Interbronchial) LN e.g.: TB or neoplastic.

3) Adventitious sounds:

A. Pleural rub:

- **Definition :** superficial leathery sound friction sound
- **Causes :** pleural diseases e.g. acute dry pleurisy
- **Timing :** inspiratory & expiratory
- **Site :** localized best heard at lower side of chest
- **Effect of cough :** unchanged by cough
- **Differential diagnosis:**
 - From friction of skin with stethoscope (Pleural rub $\uparrow\uparrow$ by firm pressure)
 - From pericardial rub (Pleural rub disappears on holding breath)
 - From other adventitious sounds (Pleural rub unchanged by forceful cough)

B. Crepitations = Crackles = Rales

C. Rhonchi (Wheezes)

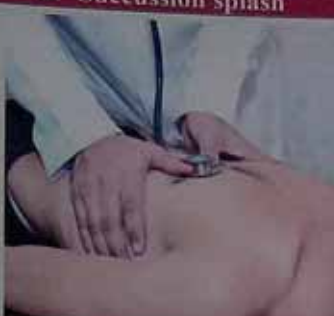
👉 In clinical exam if you heard additional sounds (as crepitations or wheezes) comment on **SACT**:

👉 **Subtype, Area (site), relation to Cough, Timing.**

- E.g. medium sized crepitations over ..., (changeable or not) on cough, inspiratory in time.
- E.g. Sonorous wheezes, generalized, not changeable on cough, expiratory in time.

Hema Adel

4) Special test:

A. Post-tussive suction	B. Succussion splash	C. Coin test
		
▪ هفضي ال cavity على الآخر يأتي ▪ أقول للعيان يكح ويبدين ياخذ نفس ▪ يقوم الهوا يخش يعلى ال cavity ▪ اسمع الشهقة اللي بعد الكحة	▪ حظ السماعة على ال chest ▪ هز العيان واسمع الطرطشة	▪ مساعد يجيب عملتين على صدر العيان ويخبط بواحد على الثانية والدكتور يسمع من ورا
❖ Heard over		
▪ Collapsible cavity "lung abscess"	▪ Hydropneumothorax	▪ Pneumothorax & big emphysematous bullae
❖ Technique of auscultation		
▪ Auscultate after prolonged cough a suction sound is heard	▪ While vigorously shaking patient a sound of turbulent fluid is heard at upper level of fluid	▪ Put coin on chest, at the same point on back put your stethoscope and strike the coin by another coin, now you listen to sound from back if you hear bell like sound it is due to pneumothorax.
D. Pneumothorax click:		
○ It is a rhythmical sound, synchronous with cardiac systole, due to left pneumothorax, ○ It is produced when there is air between the two layers of pleura overlying the heart		
E. Hamman sign: Crushing sound due to pneumomediastinum		

❖ خلاصة ال Auscultation :

1. اسمع بال diaphragm لأن معظم الدكاترة بيستخدموه وحطه firmly applied on skin
2. ابدأ بال normal healthy side
3. اطلب من العيان يتنفس من بقه والنفس يكون عميق
4. امشي كاللاتي :
 - Right side ⇔ left side
 - Aspect ⇔ aspect
 - Area ⇔ area
 - space ⇔ space
5. اسمع ال intensity of breath sound
 - وحدد هيا عالية ولا قليلة
6. اسمع نوع ال breath sound وقبل ما تقول نوعه أتأكد بال vocal resonance :
 - خلي العيان : مرة يقول 44 44 بصوت عالي ومرة يقول 44 44 بصوت منخفض او بهمس
7. أحيانا ممكن تسمع ال additional sounds :
 - اطلب من العيان ياخذ نفس جامد ويخرجه
 - اطلب من العيان يودى وشه الناحية الثانية ويكح كحيتين وكمل سمع

🏠 Chest Cases:

- COPD, Suppurative lung syndromes
- Fibrosis, Collapse, Pleural effusion

COPD	Bronchiectasis	Lung abscess	TB cavity
General exam			
- Pulse : see before ± Signs of respiratory distress; see before ± if complicated by cor-pulmonale: - Signs of RVE only epigastric pulsation felt; see before - Signs of RVF; see before - Puffiness of eyelid - MILD Clubbing of fingers. - MARKED clubbing if associated with bronchiectasis	- Recurrent fever - Toxic face, loss of weight - Puffiness of eyelid - Toxic Clubbing of fingers - Oedema of Lower limbs	Bad general condition	- SLS + TB - TB toxemia at the onset: night fever, night sweat, loss of weight, loss of appetite - Hemoptysis - Relatively good general condition
Clinically :			
Evidence of bilaterality & hyperinflation & airway obstruction	- In the basal part: signs of consolidation & may be fibrosis - In the upper parts : normal or signs of chronic bronchitis & emphysema	Signs of chronic abscess : signs of cavity	Signs of cavity & fibrosis
Inspection			
Shape & form			
Symmetrical chest: Barrel shaped chest	- Basal parts: normal shape of chest - Upper parts: barrel shaped	Normal	Unilateral Retraction on the affected side due to fibrosis
Movement: Chest expansion			
Bilaterally limitation of movements	Bilaterally limitation of movements, mainly in the basal parts	Limitation of movements on the affected side	
Palpation			
Trachea			
	Central		Shifted to same side due to fibrosis
TVF			
Decreased bilaterally all over the chest	- Basal parts: ↑ - Lower part: ↓	↑↑↑	- Cavity site: ↑ - Fibrosis site: ↓
Percussion			
- Hyper-resonance all over the chest & encroaching the normal cardiac & hepatic dullness - Ptosed liver	- Basal parts: patchy dullness - Upper parts: hyper-resonance	Dull on the affected area due to cavitation	Heterogenous dullness on the affected side
Auscultation			
Breath sounds			
- Early : vesicular breathing with prolonged expiration with <i>normal</i> intensity - Late: vesicular breathing with prolonged expiration with <i>diminished</i> intensity	- Basal parts : patchy area of bronchial breathing - Upper parts: vesicular breathing with prolonged expiration	Bronchial breathing	- Cavity site: Bronchial breathing - Fibrosis site: Diminished intensity of breath sounds
Vocal resonance			
-ve whispering pectoriloquy	+ve whispering pectoriloquy in basal parts in areas of bronchial breathing	+ve whispering pectoriloquy	Cavity site: +ve whispering pectoriloquy
Adventitious sound			
- Adventitious sound : Rhonchi : - Subtype: Polyphonic (sonorous + sibilant) - Area = site: generalized - Effect of cough: changeable but not disappear - Time: expiratory ± Inspiratory if secretion accumulated ± Crepitation : inspiratory due to secretions in recurrent chest infections	- Basal parts : medium sized or coarse Crepitations - Upper parts: rhonchi	Inspiratory medium sized or coarse Crepitations	Cavity & fibrosis site: Inspiratory medium sized Crepitations

❖ Syndrome of multiple negatives (↓ Movement, ↓ TVF, ↓ percussion note (dull), ↓ intensity of breath sounds)			
Pulmonary Fibrosis			
General exam	- Central cyanosis - Clubbing - Tachypnea ± Cor-pulmonale: Edema of LL ± respiratory failure : ± signs of respiratory distress	Collapse	Pleural effusion - Congested neck vein - Clubbing if empyema
Inspection	Unilateral Retraction on the affected side	Unilateral Retraction on the affected side	Unilateral Bulge in large effusion
Shape & form	Movement: Chest expansion: Limited on the affected side		
Palpation	Shift to same side	Shift to same side	Shift to opposite side
Trachea	TVF : ↓↓↓		
TVF	Dull on percussion		
Percussion	Heterogenous (patchy) dullness i.e. no certain topography غير منظمة	Homogenous dullness i.e. have certain topography منظمة	Basal Stony dullness
Auscultation:	Breath sounds : Decreased intensity		
Adventitious sounds	- Crepitations (inspiratory) ± rhonchi		
Vocal resonance: -ve whispering pectoriloquy			

❖ NBs:

❖ Types of fibrosis:

I. Pulmonary:

A. Parenchymatous (Unilateral): due to

↳ TB, Bronchiectasis & Lung abscess

B. Interstitial fibrosis (bilateral): idiopathic pulmonary fibrosis (Hamman Rich syndrome)

II. Pleuro-pulmonary fibrosis: fibrothorax: it may complicate hemothorax or empyema

N.B: how to deal with syndromic cases:

❖ Pickwickian syndrome = Obesity hypoventilation syndrome:

- Obesity + COPD + Cor-pulmonale

❖ Yellow nail syndrome:

- Bronchiectasis + yellow dystrophic nails + lymphedema + pleural effusion

❖ Varieties of COPD:

	A. Emphysema	B. Chronic obstructive bronchitis
Synonymous	Pink puffers = type A = emphysematous type	Blue bloaters = Type B = bronchitic type
The major abnormality	Impaired diffusion	Impaired ventilation
Leading to	Hypoxia & normocapnea	Hypoxia & hypercapnea
Dyspnea	Severe (stimulation of respiratory center due to hypoxia)	Very mild (stimulation of respiratory center; unknown reason)
Sputum	Small volume	Large volume
Central cyanosis	Absent	Present
Pulmonary HTN	Very mild or absent	Present
Cor pulmonale & RVF	Very late or absent	Present
Chest examination	Hyperinflation; barrel shaped, hyper-resonance	Wheezes; vesicular breathing with prolonged expiration
Chest x-ray	Hyper-translucency	No hyper-translucency
ABG:		
PO ₂	Mild decrease	Severe decrease
PCO ₂	Normal	high

However: there is usually overlap between both types because they frequently coexist, so it's difficult to differentiate them into 2 separate clinical types

Diagnosis Of Chest Case

❖ Etiological:	❖ Anatomical:	❖ Pathological	❖ Functional:
1) Congenital: infected cystic lung 2) Traumatic: pneumothorax 3) Neoplastic: bronchial carcinoma or adenoma 4) Inflammatory or infectious: pneumonia, COPD, TB, lung abscess. 5) Others: ○ Systemic diseases : SLE with pleurisy or interstitial fibrosis ○ Blood diseases : hemophilia or purpura with hemoptysis ○ Allergic : bronchial asthma	1) Right or Left lung or bilateral 2) Front or back 3) Upper, middle, lower zones	1) Consolidation: Pneumonia 2) Cavitation: ○ Lung abscess ○ TB cavity 3) Emphysema (+ chronic bronchitis) 4) Collapse 5) Fibrosis 6) Pleurisy 7) Pleural effusion 8) Pneumothorax 9) Hydropneumothorax	1) Compensation: ▪ Compensated or ▪ Non compensated (respiratory failure suspected by central cyanosis) 2) Complication: ❖ cor pulmonale : ▪ RVE + failure ▪ Tender pulsating liver ▪ Pulmonary hypertension ▪ TR

❖ E.g. :

- A case of COPD with bilateral emphysema , the patient is compensated but complicated with cor-pulmonale
- A case of left sided pulmonary fibrosis, mostly post tuberculous with right sided compensatory emphysema ,the patient is compensated not complicated
- A case of suppurative lung syndrome, mostly bronchiectasis, the patient is compensated but complicated with cor-pulmonale

Summary Of Chest Examination

General examination:

- Vital signs
- General overview
- Systemic overview
- Other systems except that of local exam

Local examination : Chest examination

I. Inspection

1. Shape & Symmetry
2. Chest expansion
3. Chest wall abnormality; examine the front, sides & back of the chest
4. Combined Inspection & Palpation: Respiratory Movement: Type, Rate, Rhythm & Depth
5. Combined Inspection & Palpation: Pulsations:
 - Apical, pulmonary, left parasternal & epigastric pulsations
6. Combined Inspection & Palpation: Position of mediastinum: Apex & trachea

II. Palpation

- A. Tenderness
 - B. Chest expansion
 - C. Subcostal angle
 - D. Tactile vocal fremitus (TVF)
 - E. Palpable adventitious sounds
 - 1) Anteriorly:
 - Supramammary, Mammary, Inframammary
 - 2) Laterally:
 - Upper axillary, Lower axillary
 - 3) Posteriorly :
 - Suprascapular, Interscapular, Infrascapular
- لو كان الموال يبدأ بالحاجات المشتركة ما بين ال inspection & palpation

III. Percussion

- 1) Hepatic dullness (see cardiology) & Heart percussion (see cardiology)
- 2) Chest wall:
 - A. Lung proper : Light percussion, except back by heavy percussion

- | | | |
|--|---|--|
| <ul style="list-style-type: none"> ❖ Anteriorly mainly in MCL, by light percussion: <ul style="list-style-type: none"> ○ Clavicle ○ Infraclavicular region: ○ Space by space: <ul style="list-style-type: none"> • 2nd → 6th at MCL except left 4th & 5th spaces at AAL | <ul style="list-style-type: none"> ❖ Laterally mainly in MAL, by light percussion in 4th space → 7th space | <ul style="list-style-type: none"> ❖ Posteriorly by heavy percussion: <ul style="list-style-type: none"> ○ Suprascapular (Trapezius): above spine of scapula ○ Interscapular: in paravertebral line From 2nd to 10th space ○ Infrascapular: in scapular line From 7th (scapular angle) → 10th space |
|--|---|--|

B. Special areas:

- a) Bare area of the heart: see cardiology
 - b) Kronig's Isthmus by light percussion from dullness to resonance
 - c) Traub's area by light percussion: from center to the borders
- 3) Special test: shifting dullness.

IV. Auscultation

1. Breath sounds:
 - Intensity : normal, loud, diminished
 - Type : Vesicular breathing , Vesicular breathing with prolonged expiration , Bronchial breathing & Broncho-vesicular breathing
2. Conducted sounds: Vocal resonance (Bronchophony, Whispering pectoriloquy, Aagophony)
 - Confirm bronchial breathing
3. Adventitious sounds: Pleural rub, Crepitations & Wheezes
 - Comment on SACT: Subtype, Area (site), relation to Cough, Timing
4. Special test: Post- tussive suction, Succession splash & Coin test.

SCHEME OF CHEST

I. **History:** see the algorithm of chest history for diagnosis

II. **General examination:**

- Clubbing : SLS عدم وجودها لا ينفي
- Oedema : cor-pulmonale

III. **local examination:**

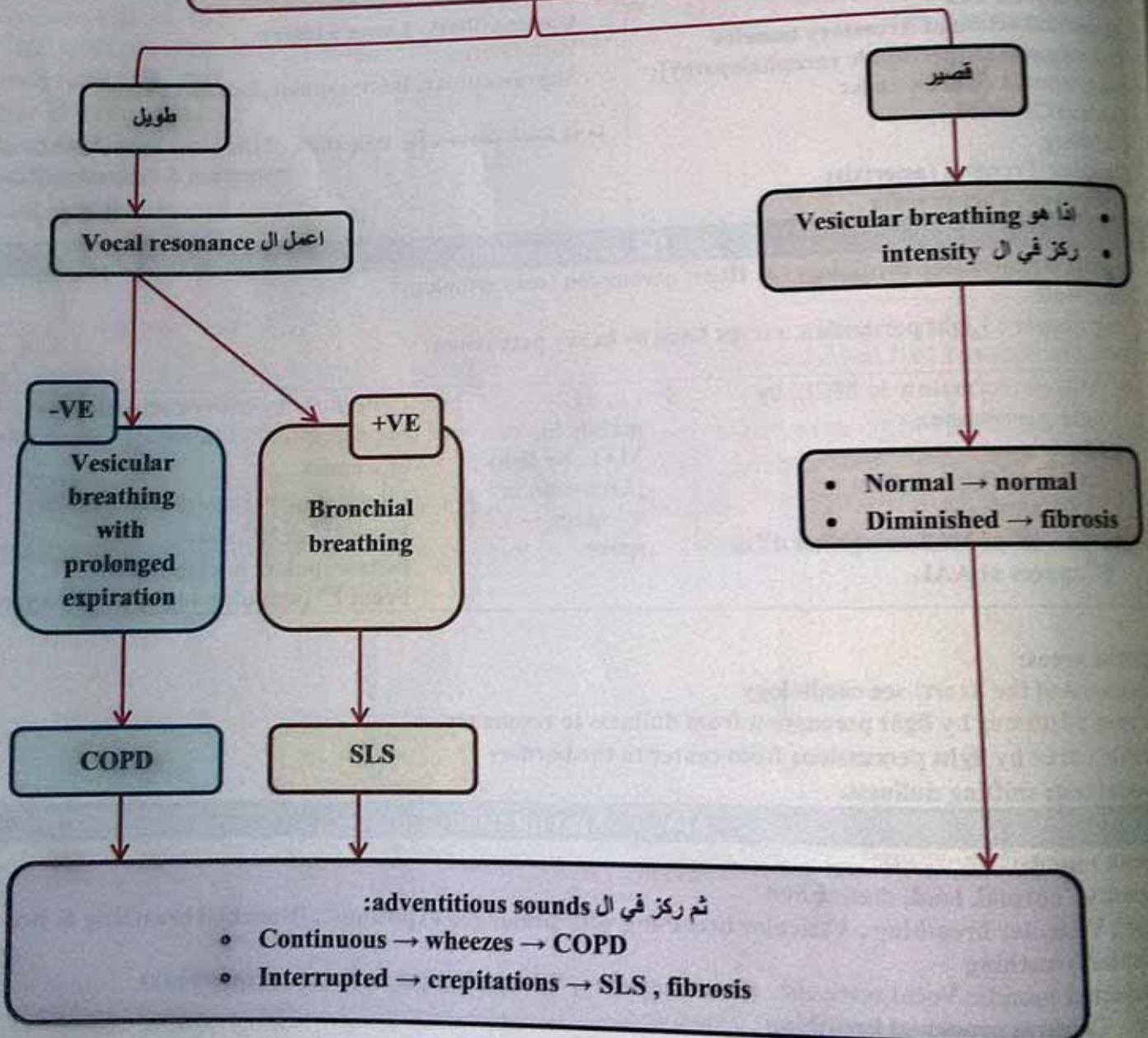
1) Chest expansion :

مفتاح ال chest عشان تعرف المنطقة المصابة

❖ Abnormally : limitation of movement detect the diseased side	
▪ Bilateral limitation	▪ Unilateral limitation
▪ In bilateral chest disease e.g. Emphysema, bronchiectasis	▪ On the affected side e.g. Fibrosis, collapse.

2) Auscultation :

- اطلب من المريض انه ينهج وهو فلتاح بقة
- ركز في طول ال expiration بالنسبة لل inspiration وحدد العلاقة:



3) Inspection , palpation , percussion : ظبطهم مع التشخيص

INVESTIGATIONS OF A CHEST CASE

I. Non-Imaging:

- 1) ECG for cor-pulmonale
- 2) Sputum analysis :
 - A. Macroscopic picture (chemical & physical properties)
 - B. Microscopic picture (cells)
 - C. Culture & sensitivity
- ❖ Indications of Sputum analysis:
 - SLS
 - May be indicated in COPD (as organism is usually known; pneumococci, haemophilus influenzae) but strongly indicated when there is failure of empirical treatment & suspected bronchiectasis
- 3) Sweat analysis in cystic fibrosis
- 4) Skin for :
 - Bronchial asthma; skin test to identify the allergen in extrinsic asthma
 - TB, tuberculin test
- 5) Serous fluid Analysis in pleural effusion to know the nature of fluid (transudate & exudate) & causative organism
- 6) Arterial blood gases (ABG):
 - ❖ According to ABG there are two types of respiratory failure :
 - Type I : hypoxia with normocapnea or hypocapnea
 - Type II : hypoxia & hypercapnea

❖ Investigation of choice:

- COPD & fibrosis → respiratory function tests
- Bronchiectasis → high resolution CT
- Lung abscess → CT & bronchoscope
- Pleural effusion → X-ray followed by aspiration
- Cor-pulmonale → ECG
- Respiratory failure → ABG

COPD: type II respiratory failure

	Normal	Respiratory failure
PO ₂	80-100 mmHg	< 60 mm Hg
PCO ₂	35 – 45 mmHg	> 50 mmHg
PH	7.35 – 7.45	↓ in type II

7) Respiratory function tests (RFTs) by spirometry:

- Forced vital capacity (FVC): it is the volume of an air maximally expired after maximum inspiration, normally : 5 liter
- Forced expiratory volume in the first second (FEV₁): it is the volume of an air maximally expired after maximum inspiration in the 1st second, normally : 4 liter
- Forced expiratory ratio (FER) = $\text{FEV}_1/\text{FVC} \times 100 = \text{normally; } 80\%$
- Residual volume (RV): it's volume of air remaining in the lung after maximum expiration, normally: 1.2 liter
- Total lung capacity (TLC): it is the volume of air that lung contain when they are fully expanded, normally 6 liter

❖ Abnormality in RFTs:

	Obstructive hypoventilation	Restrictive hypoventilation
Disease	COPD, BA	Fibrosis
RV	↑	Normal
Lung compliance	↑	↓
TLC	↑	↓
FER	↓	Normal or ↓
FVC	↓	↓



8) Bronchoscope :

❖ Diagnostic :	❖ Therapeutic :
○ Localize the site of lung abscess	○ Drain pus & inject antibiotics into cavity
○ Detect foreign body	○ Remove foreign body
○ Detect & guide biopsy of underlying malignancy	○ Inject cytotoxic drugs

9) Bedside tests to assess the respiratory function:

- Forced expiratory time : using stethoscope over trachea & calculate the time of forced expiration , Normally - 4 sec.
- Candle test or Match test : normal person can turn off a candle at 15 cm distant while his lips are widely opened
- Holding breath test: normally 10-20 sec.
- Tape test for chest expansion : see before
- Manifestation of respiratory failure: see before.

II. Imaging:

- 1) Plain X-ray for all chest diseases (AP & lateral view) especially pleural effusion
- 2) Bronchography (contrast X-ray): it WAS the investigation of choice for bronchiectasis
- 3) CT & MRI:
 - CT is the investigation of choice in lung abscess
 - High resolution CT: is the investigation of choice in Bronchiectasis
- 4) Ventilation / perfusion (V/Q) scanning : to detect suspected pulmonary embolism

→ For more details about imaging, ECG, Clinical pathology , see Paraclinical Made easy for the author.

CLINICAL DISCUSSION

❖ What is the difference between Littin's sign & Litten's sign ?

- Littin's sign: see before in the signs of respiratory distress (see before)
- To understand the Litten's signs , we should discuss first the Litten's phenomenon:

⊕ Litten's Phenomenon:

- Normally a rippling shadow moving down the lower intercostal space is seen following diaphragmatic descent during deep inspiration. This is visible only in very thin individuals with a bright light applied obliquely to the chest wall (this shouldn't be mistaken with normal inspiratory retractions of lower intercostal space; Littin's sign)

⊕ Litten's sign:

- Definition : unilateral loss of litten's phenomenon i.e. unilateral loss of rippling shadow (compared to the other side)
- Mechanism : unilateral interference of normal diaphragmatic descent
- Causes :
 - Unilateral phrenic nerve palsy e.g. diaphragmatic paralysis (diaphragmatic)
 - Unilateral severe lower lobar disease (supra- diaphragmatic)
 - Unilateral severe subphrenic disease (infra- diaphragmatic)

❖ How can you differentiate between crepitations & pleural rub ?

	Crepitations	Pleural rub
Firm pressure by stethoscope	No change	↑↑↑
Effect of cough	May be affected	Never affected
Time	Mainly inspiratory	Inspiratory & expiratory
Site	Profuse	Localized

? How to know site of shifted trachea by auscultation عند الطلب و السؤال عليها فقط

الطبيعي تسمع bronchial breathing على ال sternomastoids
 حط السماعة على ال 2 sternomastoid ولاحظ علو الصوت فين .
 ال shifted trachea تبقى للناحية اللي الصوت فيها أعلى

❖ What are the differences between bronchiectasis and bronchiectasis sicca ?

	Bronchiectasis	Bronchiectasis "sicca hemorrhagica"
▪ Cause	Any infection causing weakness of bronchial wall	Secondary to TB
▪ Site	Basal	Apical
▪ Sputum	Excessive expectoration	Dry = sicca = no sputum
▪ Blood in sputum	Blood tinged sputum	Frank hemoptysis = hemorrhagica
▪ Prognosis	Good prognosis	Bad prognosis

❖ How to differentiate between pulmonary and pleural causes as a cause of dullness over the base of left lung?

• By percussion over Traube's area

▪ Pleural origin : dull Traube's area	▪ Pulmonary origin : resonant Traube's area
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❖ What are the differences between wheezes & rhonchi ?

Wheezes	Rhonchi
Symptom	Sign
High pitched	Low pitched
Heard at the periphery	Heard in the center
Not palpable	Palpable

❖ Which have the higher possibility of risk, generalized rhonchi or localized rhonchi ?

• Localized rhonchi because it may be tumor

❖ How to suspect tumor lesion from auscultation ?

• Hearing rhonchi with the following characters :

▪ Monophonic (tumour obstruct one bronchus)	▪ Localized	▪ Fixed with cough
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❖ How to differentiate between dyspnea due to heart disease & chest disease ?

Cardiac dyspnea	Respiratory dyspnea
❖ Time	
○ Nocturnal (PND; 1-2 hours after sleep) and associated with orthopnea	○ Early in the morning
❖ Associated symptoms	
○ Palpitation, syncope, angina pain, hypertension	○ Fever, sweating, wheezes
❖ Examination	
○ Chest: generalized wheezes, bilateral basal crepitations	○ Chest : generalized wheezes
○ Heart: gallop & murmur	○ Heart: normal

❖ Why are bronchial asthmatic rhonchi peripheral mainly while that of chronic bronchitis is central mainly?

• Because bronchial stenosis due to **bronchial asthma** is based on bronchospasm which needs smooth muscles which lie more in the **peripheral** bronchi

• While bronchial stenosis of chronic bronchitis is based on mucosal edema, hypertrophy & mucosal hypersecretion which needs bronchial mucosa which lie more in the central bronchi

❖ How to suspect diaphragmatic paralysis by examination?

- 1) Reversed tidal percussion
 - 2) Litten's sign
 - 3) Absent litten's sign
 - 4) Abdominal paradox : abdominal retraction during inspiration
 - 5) Thoracic respiration
 - 6) Shifting of apical pulsation upwards
 - 7) Right lung base dullness
 - 8) Abdominal alternans: in diaphragmatic paresis (not paralysis), periods of normal abdominal movement alternating with absent abdominal movement
- N.B: However **definitive diagnosis** by paradoxical movement in fluoroscopy (x-ray + fluorescent screen → moving images of the internal structures)

❖ What are the complications of COPD ?

• Respiratory failure • Right sided heart failure following cor-pulmonale • Left sided heart failure	• Pulmonary infections • Pneumothorax • Bronchiectasis	• Polycythemia • Salt & water retention • Complication of chronic cough
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❖ What are the causes of left ventricular failure in COPD?

- Associated disease of the left side of the heart especially coronary artery disease
- Reversed Bernheim effect
- Acidosis leading to myocardial depression
- Hypoxia leading to myocardial ischemia
- **Volume overload** due to hyperdynamic circulation due to vasodilatation
- **Increased blood viscosity** due to polycythemia

❖ What is the value of sputum examination in chest case?

1. **Amount /24h** : big (> 1 cup/day) in suppurative lung syndrome
2. **Postural variation**:
 - Lung abscess: ↑ by lying on healthy side
 - Bronchiectasis : ↑ by stooping (leaning) forward
3. **Colour** :
 - White : chronic bronchitis
 - **Green**: pyocyanous , anaerobic infection
 - **Yellow** : pyogenic infection
 - **Black**: cigarettes smokers, industrial cities
4. **Character** : mnemonic; **FOR WOMAN**
 - Frothy : acute pulmonary edema
 - Red current jelly : bronchial Carcinoma, Friedlander's pneumonia
 - Rusty : altered blood in pneumonia
 - Watery : hydatid cyst, alveolar cell carcinoma
 - Mucoid (tenacious) or mucus pellet: chronic bronchitis & bronchial asthma
 - Mucopurulent or purulent : bronchitis, bronchiectasis, SLS
 - Anchovy sauce "Chocolate" : amebic lung abscess
 - Nammular "coin like" : caseous material in TB
5. **Diurnal variation**:
 - Nocturnal: heart
 - Early morning : bronchiectasis & bronchial asthma
6. **Seasonal variation** :
 - Bronchiectasis : winter exacerbation
7. **Odour** : what are the causes of Fetid sputum ?
 - Foul fetid : suppurative lung syndrome & infection by anaerobes , spirochetes, fusiform bacilli & fungi

❖ What is the differential diagnosis of dullness in 2nd left space ?

- 1) Chest wall: e.g. Tumor
- 2) Pleura: apical pleural thickening, massive pleural effusion
- 3) Left lung :
 - Consolidation of left upper lobe, specially due to TB
 - Collapse of left upper lobe
 - Fibrosis of left upper lobe
 - Pancoast tumor
- 4) Heart:
 - Pulmonary artery dilatation, pericardial effusion
- 5) Superior mediastinum : thymus, thyroid & dermoid cyst
- 6) Abdomen : ascites & pregnancy - these will raise diaphragm & subsequently heart

❖ What are the main causes of cough ? (الإجابة بالترتيب ...)

1. Chronic bronchitis, The most common cause	2. Suppurative lung syndromes	3. TB
4. Bronchogenic carcinoma	5. Chronic sinusitis (post nasal discharge)	

❖ What are the complications of cough?

A. Thoracic complications :			
▪ Intercostal myalgia (muscle pain) ▪ Rib fracture (stress fracture)	▪ Spontaneous pneumothorax	▪ Hemoptysis	▪ Emphysema ▪ Chronic bronchitis
B. Extrathoracic complications:			
▪ Abdominal : hernia , stress incontinence , rectal or uterine prolapse		▪ Head : cerebral hemorrhage & subconjunctival hemorrhage	
C. General : insomnia			

❖ What are the causes of acute dyspnea?

• Acute massive myocardial infarction	• Acute massive lung collapse	• Acute pulmonary embolism	• Tension pneumothorax
• Pneumonia , pleurisy	• Bronchial asthma	• Hysterical	

❖ What are the causes of paroxysmal dyspnea?

❖ Paroxysmal dyspnea with wheezing	❖ Paroxysmal dyspnea without wheezing
• Bronchial asthma • Cardiac asthma • Uremic asthma • Carcinoid syndrome • Pulmonary vasculitis	• Cardiac dyspnea • Extrinsic allergic alveolitis • Tetany (laryngismus stridulus) • Myasthenic crisis • Recurrent pulmonary emboli

❖ What are the causes of dyspnea at rest ?

• Severe degree of exertional dyspnea	• Postural dyspnea	• Paroxysmal dyspnea
• Acute dyspnea	• Psychogenic dyspnea	

❖ What are the causes of cor pulmonale ?

A. Acute cor pulmonale :	B. Subacute cor pulmonale:	C. Chronic cor pulmonale :
▪ Massive pulmonary embolism ▪ Tension pneumothorax ▪ Massive lung collapse	• Lymphangitis carcinomatosa • Recurrent showers of pulmonary emboli	• Hypoxic cor pulmonale: occurs in almost cases of chronic lung diseases e.g. COPD & interstitial lung disease • Vascular cor pulmonale : primary pulmonary hypertension & pulmonary bilharziasis

❖ What are the pulmonary causes of chest pain ?

A. Respiratory	B. Mediastinal	C. Chest wall
• Acute pleurisy : primary or secondary to lung disease (pneumonia): stitching, referred to abdominal wall (T6-T11) shoulder (C3, C4), arm (C8- T1 in apical pleurisy) • Pleural effusion : dull aching pain • Pneumothorax: acute tearing chest pain, • Acute massive lung collapse • Pulmonary infarction : acute pain + hemoptysis + dyspnea • Lung diseases extending to pleura	• Mediastinal tumors • Esophageal : spasm reflux esophagitis, cancer	• Skin : infection, tumor • Breast : mastitis, abscess, tumor • Ribs : fracture, osteomyelitis • Intercostal muscles : muscle strain, myositis • Intercostal N.: root pain, Herpes Zoster

❖ What are the types of hemoptysis?

A. True :		B. Spurious (false):	
▪ Blood originating from below vocal cords		▪ Blood originating from above vocal cords	
Laryngoscope ← إزاي تفرق ما بين النوعين ؟		▪ Blood is on surface of sputum	
❖ Causes : ○ Frothy blood tinged : acute pulmonary oedema ○ Blood stained (blood + sputum) : البلم فيه نقط دم : acute bronchitis (the most common cause) & bronchogenic carcinoma ○ Blood streaked (blood streaks + sputum) : البلم معرق بدم : pulmonary TB, chronic bronchitis , bronchogenic carcinoma ○ Frank blood دم صافي : pulmonary TB, bronchial adenoma & carcinoma, bronchiectasis & lung abscess & pulmonary infarction , Mitral stenosis ○ Red current jelly (blood + mucous + lung debris): bronchial carcinoma ○ Rusty: red brown sputum (altered blood pigment): lobar pneumonia		❖ Causes : ○ Bleeding gums (scurvy) ○ Nasopharynx (inflammation, tumors, ulcers)	

❖ What is the treatment of bronchial asthma ?

❖ Between attacks :	❖ During attacks:
• Avoid irritating agent • Oral corticosteroids • Mast cell stabilizers (intal, ketofen)	• O2 therapy • Bronchodilators (aminophylline (IV), salbutomal (inhaler) • Mucolytic • Antibiotics

O.S.C.E QUESTIONS

• Inspect, palpate, percuss & auscultate chest	• Percuss special areas of the chest
• Detect signs of hyperinflation by inspection, palpation & percussion	• Signs of COPD in the face
• Examine the lower lobes of the lung	• Examine the middle lobe of the lung
• Examine the apices of both lower lung lobes	
• General examination related to the chest	

FREQUENTLY ASKED QUESTIONS FOR MID & END ROUND EXAM

Test yourself!

1) What is the surface anatomy of right lung?	2) What is the surface anatomy of left lung?
3) What is the surface anatomy of oblique fissure?	4) What is the interscapular region formed of?
5) Examine lower lobes equal what?	6) What are chest causes of jaundice?
7) What are chest causes of cyanosis?	8) What are chest causes of clubbing?
9) What are chest causes of pallor ?	10) What are the causes of edema LL?
11) What is the importance of pulse examination in a chest case?	12) What are the causes of suppurative lung syndrome?
13) Describe Barrel chest?	14) What is the causes of barrel cheat?
15) What are the causes of symmetrical chest shape?	16) How to differentiate normal from abnormal hemithorax (bulge & retraction) clinically?
17) What are the causes of unilateral chest bulge	18) What are the causes of abdominal respiratory movement?
19) What are the causes of thoracic respiratory movement ?	20) What is the normal respiratory rate?
21) What are causes of bradypnea?	22) What are the causes of rapid deep breathing?
23) What are the causes of rapid shallow breathing?	24) What are accessory muscles of inspiration?
25) What is the aim of pursing lips?	26) What is Litten's sign?
27) Importance of examination of cardiac pulsation in chest patient?	28) Causes of shifted trachea?
29) Causes of tender chest wall?	30) Causes of increased TVF?
31) Causes of decreased TVF?	32) Causes of palpable rhonchi?
33) Causes of limited chest expansion?	34) Trail's sign?
35) Cause of dullness on long?	36) Cause of stony dullness on lung?
37)	38) Surface anatomy of Krong's isthmus?
39) Causes of dullness on Kronig's isthmus?	40) Borders & Surface anatomy of Traub's area?
41) Content of Traub's area?	42) Note of percussion of normal Traub's area?
43) Physiological causes of dullness on Traub's area?	44) How to differentiate between pulmonary and pleural causes as a cause of dullness over the base of left lung?
45) How to diagnose hydropneumothorax by percussion?	46) Causes of diminished intensity of breath sounds?
47) Describe normal vesicular breathing?	48) Describe characters of vesicular breathing with prolonged expiration?
49) Causes of vesicular breathing with prolonged expiration?	50) Describe bronchial breathing?
51) Causes of bronchial breathing?	52) What is the classification of bronchial breathing?
53) What is the significance of the pitch of rhonchi?	54) What is the significance of the timing of rhonchi?
55) What is the significance of the distribution of rhonchi?	56) What is the significance of the cough on rhonchi?
57) What are causes of fine (or late inspiratory) crepitations ?	58) What are causes of coarse or early inspiratory crepitations?
59) What are the causes of pleural rub?	60) D'Espin sign?
61) What are special tests in chest auscultation ?	62) What is meant by vocal resonance?
63) What are the investigations of chest case?	64) What are the manifestation of respiratory failure?
65) What are the causes of Cor-pulmonale?	66) What are the manifestation of cor-pulmonale?
67) What is treatment of bronchial asthma ?	68) What are the complications of COPD?
69) Why are bronchial asthmatic rhonchi peripheral mainly while that of chronic bronchitis is central mainly?	70) How to differentiate between hemoptysis & hematemesis?
71) Definition & causes of tracheal tug?	72) Pulmonary function test?
73) Define emphysema?	74) Succussion splash ?
75) Coin test?	76) Causes of acute chest pain ?
77) All questions answered in the clinical discussion are very important.	

Chronic Obstructive pulmonary Disease (COPD)

❖ Personal history

❖ **Complaint:** Shortness of breath 3 days duration.

❖ HPI:

- The condition started 50 years ago by paroxysmal attacks of **cough**, **dyspnea** and **wheeze** of gradual onset and progressive course. These attacks were precipitated by exertion and dust exposure, relieved by rest and medication. The patient was completely free in between attacks.
- 20 years later, **dyspnea** progressively increased till became at rest with no orthopnea or PND and **wheeze** became more continuous even in between attacks and the patient developed **productive cough**. The sputum was small amount, less than one cup per day, **whitish** in colour, **mucoid** in character, **odourless**, increased in the morning with **no relation to certain posture**.
- The patient sought medical advice, investigated by CXR and sputum analysis, treated by inhaler and mucolytics and was advised to stop smoking.
- The patient remained symptoms-free till 3 days ago when he experienced another similar attack of **dyspnea** at rest with **cough** with mucoid whitish odourless **sputum** and continuous **wheeze**.
- No Symptoms of **systemic congestion**,
- No **cyanosis**,
- No **haemoptysis**,
- No **Chest pain**,
- No **Constitutional Symptoms**,
- No Symptoms suggesting **other system** affection.

❖ Past history

- No History of Drug intake.
- No History of DM, HPN, T.B.
- No History of admission to chest sanatorium.
- No history of operations.

❖ Family history

- No consanguinity.
- No similar condition in family.
- No common disease in family.

1. General examination:

- The patient is fully **conscious**, well oriented for time, place and person. Average mood and memory. The patient is co-operative with average intelligence.
- **Temperature:** 37° c.
- **Bl. Pressure:** 150/60.
- **Resp. Rate:** 16/minute, regular, average depth, abdomino-thoracic.
- **Pulse:** Regular, 60 beat/minute, **big pulse volume**, **water hammer pulse**, equal on both sides, intact peripheral pulsation, vessel wall is not felt.
- **The patient** looks well, average built, **no cyanosis**, pallor or jaundice. He is lying free flat comfortable in bed.
- **Head & Neck:** puffy eyelids, arcus senilis, nicotine stain on his lips with no sub-conjunctival hemorrhage. Working ala nasi or pursing of lips. Neck veins are pulsating **not congested**.
- **Upper limb:** fine tremors (probably senile or due to β stimulant but **no flapping tremors**), **No clubbing**.
- **Lower Limb:** mild bilateral pitting painless **edema** reaching mid leg level.

2. Local Examination:

a) Inspection

- **Chest Wall:** some brownish pigmentation, with no scars, no dilated veins.
- **Resp. Movement:**
 - **Rate:** 16/minute, regular, average depth, abdomino-thoracic.
 - **Expansion:** bilateral limitation in chest expansion.
 - **Signs of action of accessory Muscles of respiration:** there is suction of supraclavicular fossa, inspiratory indrawing of lower intercostals spaces with no visible contraction of sternomastoid or elevation of thoracic cage (Signs of action of accessory muscles of inspiration). The patient is not pursing his lips or grasping a chair (Signs of action of accessory muscles of expiration). No Hoover's sign or tracheal tug (Signs of low flat diaphragm).
- **Shape of the chest:** symmetrical, Barrel shaped chest. ($\uparrow$ AP diameter = Tr. Diameter, raised shoulder, obtuse subcostal angle).
- **Mediastinum:** Central trachea, No trill's sign, No tracheal Tug with **Absent Apex**.
- **Pulsations:** Apex is not visible. There is visible epigastric pulsations (probably aortic).

b) Palpation:

- **Bilateral limitation** in chest expansion.
- **Central Trachea** with **decreased tracheal length** but **no tracheal tug**.
- **Absent Apex** but there is **epigastric pulsations** (Probably Aortic in origin $\rightarrow$ HDC).
- **TVF:** equal on both sides.
- **No palpable Rhonci** or rub.
- **No chest wall tenderness.**

c) Percussion:

- **Hepatic dullness** at **Rt. 6th Space MCL** with **normal hepatic span** (=14cm) indicating **encroachment on hepatic dullness**.
- **Heart:** No dullness to the right border of sternum, both aortic and pulmonary areas are resonant, preserved waist of the heart, no dullness outside the apex, lower end of the sternum is impaired note with **resonant bare area** of the heart (cardiac dullness is encroached upon by hyper-inflated lung).
- **Lung:** generalized hyper-resonance with encroachment on hepatic and cardiac dullness:
 - **Front:** Resonant clavicles, infra-clavicular areas, dullness is detected at 7th space Rt. and Lt. MCL.
 - **Lateral:** Dullness at 9th space Rt. And Lt. MAL.
 - **Back:** Dullness at 11th space Rt. And Lt. SL.
 - **Resonant Bare area**, Resonant **Kronig's isthmus**.
 - **By Tidal Percussion** $\rightarrow$ Diaphragm is freely mobile.
 - **No Shifting dullness.**

d) Auscultation:

- Diminished vesicular breath sound with prolonged expiration and diminished V.R. Associated with generalized polyphonic expiratory and inspiratory rhonci changeable with cough.

3. Other systems examination:

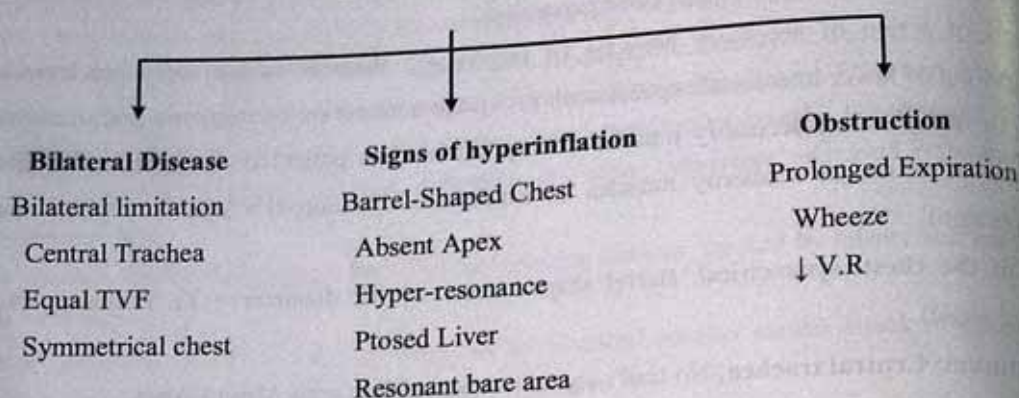
- Enlarged liver with no tenderness.

? What is your diagnosis ?

☐ A Case of COPD mostly Allergic (chronic Asthmatic bronchitis), The patient is compensated not complicated

? Why COPD :

☐ Combination of:



? Why Allergic, why chronic bronchitis ??

- History of asthmatic attacks (with dyspnea) on exposure to allergens (asthma).
- History of heavy smoking (chronic bronchitis).

? Why compensated/not Complicated ?? → No Cor Pulmonale or Respiratory Failure.

► No Cor Pulmonale ::

- From History: No systemic congestion.
- Examination: No neck veins, LL edema (Can be explained due to CO₂ retention or use of steroids), No tender hepatomegaly, No signs of Rt. V. hypertrophy.

► No Respiratory Failure::

- History: No cyanosis.
- Clinically: No tremors, cyanosis or disturbed consciousness.
- Lab. : ABG (The most important as RF is a lab. Diagnosis).

? How would you proceed to manage this case?

► Investigation

For etiology: CBC, ↑ IgE, Skin tests.

For Functional Diagnosis: ABG, ECG, CXR, ECHO

For main diagnosis: Sputum analysis, CXR, Pulmonary Function Test (Of Choice).

► Treatment

- Avoid Exposure to irritation.
- TTT of pulmonary infections.
- Symptomatic ttt: expectorant, mucolytic, bronchodilator, steroids.
- TTT of complications: Resp. failure & Heart failure, polycythemia, bullae.

Cor-pulmonale

❖ Differs from COPD in:

1- From History : Symptoms of systemic congestion.

2- Examination :

- Congested neck veins with expiratory filling, Bilateral pitting L.L edema & Enlarged tender liver in abdominal exam.

→ Otherwise, It's the same.

Fibrosis

❖ Personal history

❖ **Complaint:** Shortness of breath 1 month duration.

❖ HPI:

- In a known diabetic patient, The condition started 15 years ago by acute onset, progressive course of **night fever**, **night sweat** which was associated with dry **cough** with no special character, no postural, seasonal or diurnal variation. 4 days later, he experienced stitching right sided **chest pain** which was localized, increased with cough and inspiration, relieved by rest, holding breath, lying on Rt. Side, not related to exertion. It was associated with gradual onset and progressive course of **dyspnea at rest** with no orthopnea or PND. The patient was admitted to Ain-Shams University Hospital, investigated by CXR, pleural fluid analysis and was diagnosed as massive pleural effusion. He was treated by antibiotics, aspiration of 1.5 liters of turbid yellowish fluid with no complications.
- The patient remained symptoms-free for one and half month, then he re-experienced similar attack of **dry cough**, **dyspnea at rest** and right sided localized stitching **chest pain**. The patient was re-admitted to Ain-Shams University Hospital, re-investigated by CXR, CBC, Tuberculin test which was positive. The patient was treated by aspiration of yellowish turbid fluid and he received medications in the form of tablets, capsules and injections. He was advised to follow this regimen for one year and he completed the course of treatment.
- The patient remained quite well till one month ago when he developed gradual onset, progressive course of **dyspnea** on less than ordinary effort, relieved by rest with no orthopnea, no PND.
- No Symptoms of **systemic congestion**.
- No **cyanosis**, No **haemoptysis**.
- No **pressure** symptoms.
- No nocturnal diarrhea, no palpitation, no unsteadiness with sudden standing, no gustatory sweating, no impotence (**symptoms of autonomic neuropathy**)

❖ Past history

- Past history of D.M. since he was 10 years, manifested by polyuria, polydipsia, polyphagia, investigated by fasting blood sugar, and treated with insulin for life.
- No History of Drug intake.
- No History of HPN.
- No history of operations.

❖ Family history

- No consanguinity.
- No similar condition in family.
- No common disease in family.

1. ▶ General exam

- The patient is fully **conscious**, well oriented for time, place and person. Average mood and memory. The patient is co-operative with average intelligence.
- **Temperature:** 37.2° c.
- **Bl. Pressure:** 130/80 (in both standing and recumbent position).
- **Resp. Rate:** 16/minute, regular, average depth, abdomino-thoracic.
- **Pulse:** Regular, 60 beat/minute, average volume, no special character, equal on both sides, intact peripheral pulsation, vessel wall is not felt, no radio-femoral delay
- **The patient** looks well, average built, **no cyanosis**, pallor or jaundice. He is lying free flat comfortable in bed.
- **Head & Neck:** nicotine stain on his lips with no sub-conjunctival hemorrhage, no working ala nasi or pursing of lips. Neck veins are pulsating **not congested**.
- **Upper limb:** No flapping tremors, **No clubbing**.
- **Lower Limb:** No L.L. **edema**, with some scars (traumatic), some trophic changes, diabetic dermopathy.

2. Local Examination:

A. Inspection:

- **Chest Wall:** No scars, no dilated veins, no pigmentation.
- **Resp. Movement:**
 - **Rate:** 16/minute, regular, average depth, abdomino-thoracic.
 - **Expansion:** Limitation in chest expansion on the Rt. Side.
 - **Signs of action of accessory Muscles of respiration:** No suction of supraclavicular fossa, no inspiratory indrawing of lower intercostal spaces with no visible contraction of sternomastoid or elevation of thoracic cage (No working accessory muscles of inspiration). The patient is not pursing his lips or grasping a chair (Signs of action of accessory muscles of expiration). No Hoover's sign or tracheal tug (Signs of low flat diaphragm).
- **Shape of the chest:** asymmetrical, Retraction on Rt. Side, acute subcostal angle.
- **Mediastinum:** shifted **trachea** to the Rt. side (trail's sign), No tracheal Tug, **the Apex** is in Lt. 5 space MCL.
- **Pulsations:** There is visible epigastric pulsations (probably aortic).

B. Palpation:

- **Limitation** in chest **expansion** on Rt. Side.
- **Central Trachea** with **decreased tracheal length** but **no tracheal tug**.
- **Apex** is in the left 5th space MCL & **epigastric pulsations** (Probably Aortic in origin).
- **TVF:** increased on Rt. Side especially at apical parts!!!! *(it is supposed it is decreased but may be attributed in this case to tracheal shift to the right side).*
- No **palpable Rhonci** or rub.
- No chest wall **tenderness**.

C. Percussion:

- **Hepatic dullness** at Rt. 5th Space MCL.
- **Heart:** impaired note to the right border of sternum, both aortic and pulmonary areas are resonant, preserved waist of the heart, no dullness outside the apex, lower end of the sternum is impaired note.
- **Lung:**
 - **Front:** Resonant clavicles, infra-clavicular areas, Heterogeneous dullness is detected from 3rd space downwards in Rt. MCL.
 - **Lateral:** Heterogeneous dullness at 4th space downwards in Rt. MAL.
 - **Back:** Heterogeneous dullness at Rt. SL.
 - **Bare area:** impaired note, Resonant **Kronig's** isthmus.
 - By **Tidal Percussion** → Diaphragm is freely mobile.
 - No **Shifting dullness**.

D. Auscultation:

- Normal breath sound on Lt. side, diminished air entry on Rt. Side with decreased V.R with pan inspiratory fine crepitations not changed with cough at Rt. Infra-mammary area.

3. Other system examination:

See neurology for Diabetic Peripheral neuropathy (P.N) *(All signs of diabetic peripheral neuropathy are present in this case)*

- A Case of Right Sided pleuro-pulmonary fibrosis secondary to T.B. The patient is compensated, complicated w/hy Fibrosis ??

On the Rt. Side there is:

1. ↓ Expansion by inspection.
2. ↓ TVF by palpation.
3. ↓ Resonance by percussion.
4. ↓ Breath sound by auscultation.

So, It's a syndrome of multiple negatives either:

- Pleural Effusion (excluded by retraction and mediastinal shift to Rt.)
- Collapse } D.D by percussion → Homogenous
- Fibrosis } → Heterogeneous

? **Why Rt. sided ?** from History (site of pain & aspiration) and examination.

? **Why Secondary to T.B ?** from History (T.B toxemia) and from Tuberculin test.

? **Why compensated/not Complicated?** → No Cor Pulmonale or Respiratory Failure.

► **No Cor Pulmonale ::**

- From History: No systemic congestion.
- Examination: No neck veins, L.L edema, No tender hepatomegaly, No signs of Rt. V. hypertrophy.

► No R.F ::

- **History:** No cyanosis.
- **Clinically:** No flappy tremors ,no cyanosis or disturbed consciousness.
- **Lab. :** ABG (The most important as RF is a lab. Diagnosis).

? What are the causes of upper zone lung fibrosis?

- TB
- Allergic bronchopulmonary aspergillosis
- Ankylosing spondylitis
- Asbestosis

? How would you proceed to manage this case?

❖ Investigation

- For etiology: Tuberculin test, CBC, ESR.
- For Functional Diagnosis: ABG, ECG, CXR, ECHO
- For main diagnosis: Pleural fluid analysis, CXR, Biopsy, Pulmonary Function Test.

◆ Treatment

- Control of diabetes and treatment of peripheral neuropathy (see neuro)
- Treatment of T.B (if still active).
- Supportive ttt for fibrosis

TB cavity versus Lung abscess

❖ **Personal history...**

❖ **Complaint :** Shortness of breath of one day duration.

❖ **HPI:**

- The condition started 15 years ago by gradual onset and progressive course of **productive cough**, the sputum was **small** in amount, **thick, whitish, odorless and not related to posture**. One week later, he experienced **night fever**, **night sweat, loss of appetite and loss of weight**. He was admitted to El-Abbasia Chest Hospital, investigated by CXR, tuberculin test, sputum analysis and culture and diagnosed as **pulmonary T.B**. He received anti-tuberculous treatment for 2 month at hospital and was advised to continue this regimen for 7 more months at home but unfortunately, he discontinued the treatment after one month.
- One month after he stopped anti T.B treatment, he developed **productive cough** of **yellowish, fetid sputum**, **1 cup in amount, increased by sleeping on the right side** and in the **early morning** associated with **exertional dyspnea** on ordinary effort (i.e on climbing the 2nd floor) not associated with orthopnea or PND associated with **continuous wheeze**. He was re-admitted to El-Abbasia Chest Hospital, investigated by CXR, tuberculin test, sputum analysis and culture, treated by mucolytic, expectorants and inhaler and was advised to stop smoking.
- The patient was quite well till one day ago when he developed gradual onset and progressive course of **exertional dyspnea** on less than ordinary effort with no orthopnea or PND.
- No Symptoms of **systemic congestion**.
- No **cyanosis**, No **haemoptysis**.
- No **Chest pain**.
- No **Pressure symptoms**.
- No **Constitutional Symptoms**.
- No Symptoms suggesting **other system** affection.
- ❖ **Past history:**
 - No History of DM, HPN.
 - No History of admission to chest sanatorium.
 - No history of drugs or operations.
- ❖ **Family history:**
 - No consanguinity.
 - No similar condition in family.
 - No common disease in family

1. **General examination :**

- The patient is fully **conscious**, well oriented for time, place and person. Average mood and memory. The patient is co-operative with average intelligence.
- **Temperature:** 37° c.
- **BL Pressure:** 120/70.
- **Resp. Rate:** 16/minute, regular, average depth, abdomino-thoracic.
- **Pulse:** Regular, 60 beat/minute, average volume, no special character, equal on both sides, intact peripheral pulsation, vessel wall is not felt.
- **The patient** looks well, average built, **no cyanosis**, pallor or jaundice. He is lying free flat comfortable in bed.
- **Head & Neck:** no puffiness in eyelids, no sub-conjunctival hemorrhage, no working ala nasi or pursing of lips. Neck veins are pulsating **not congested**.
- **Upper limb:** No **clubbing**, no **flapping tremors**.
- **Lower Limb:** no L.L edema.

2. Local examination:

A. Inspection:

- **Chest Wall:** no scars, no dilated veins, no pigmentation.
- **Resp. Movement:**
 - **Rate:** 16/minute, regular, average depth, abdomino-thoracic.
 - **Expansion:** limitation in chest expansion on Lt. side.
 - **Signs of action of accessory Muscles of respiration:** there is no suction of supraclavicular fossa, no inspiratory indrawing of lower intercostals spaces with no visible contraction of sternomastoid or elevation of thoracic cage (Signs of action of accessory muscles of inspiration). The patient is not pursing his lips or grasping a chair (Signs of action of accessory muscles of expiration). No hoover's sign or tracheal tug (Signs of low flat diaphragm).
 - **Shape of the chest:** retraction on the lt. side with normal form.
 - **Mediastinum:** Central trachea i.e No trail's sign, No tracheal Tug with visible apical pulsation.
 - **Pulsation:** visible apical and epigastric pulsation (Normal findings).

B. Palpation:

- **Limitation** in chest **expansion** on Lt. side.
- **Central Trachea** (*may be shifted in these cases*) with **no tracheal tug**.
- **Apex:** Regular, 70/min., in the left 5th space MCL, localized, normal character, no thrill and no rocking movement.
- **TVF:** increased in the Lt. supra-mammary area and decreased in the Lt. infra-mammary area.
- No **palpable Rhonci** or rub.
- No chest wall **tenderness**.

C. Percussion:

- **Hepatic dullness** at Rt. 5th Space MCL.
- **Heart:** No dullness to the right border of sternum, aortic area is resonant with mild dullness in pulmonary area, dullness in left 3rd space (waist), dullness outside the apex, lower end of the sternum is dull (*fibrosis*), with dull bare area.
- **Lung:** dullness in the lower part of Lt. lung.
 - **Front:** Resonant clavicles, infra-clavicular areas, slight dullness is detected in left 2nd, 3rd and 4th (AXL) spaces with stony dullness in left 5th (AXL) and 6th at MCL.
 - **Lateral:** Dullness in the left 7th and 8th spaces at MAL.
 - **Back:** Dullness in left 9th and 10th at SL.
 - Dull **Bare area**, Resonant **Kronig's isthmus**.
 - By **Tidal Percussion** → Diaphragm is freely mobile.
 - No **Shifting dullness**.

D. Auscultation :

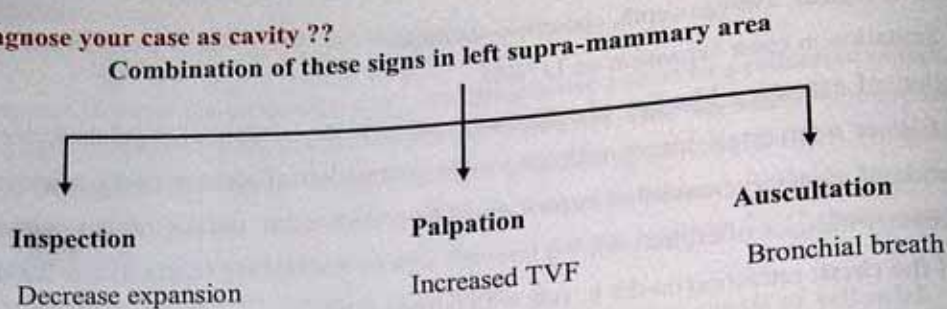
- **Bronchial breathing** with +ve V.R. in left supra-mammary with **diminished air entry** in the Lt. infra-mammary areas (Vesicular breath sound with prolonged expiration with diminished vocal resonance and expiratory polyphonic rhonchi can be auscultated on the right side as there is underlying chronic bronchitis as he is chronic heavy smoker).

3. Other systems examination...

? What is your diagnosis ?

A Case of Left sided tuberculous apical cavity with basal fibrosis, The patient is compensated not complicated.

? Why did you diagnose your case as cavity ??



? Why did you diagnose your case as TB cavity not a lung abscess ??

It is TB because:

By history:

- There is history suggestive of TB toxemia at the onset: night fever etc...
- There is history of hemoptysis which is so common in TB.
- There is history of admission to a TB sanatorium.
- There is history of receiving drugs for long period (anti-TB drugs).

By examination: (signs of fibrosis + signs of cavitation)

→ There are clinical signs more apparent in the upper lung field (left supramammary) in the form of cavitation & fibrosis on the left side by:

→ By inspection:

- Shape: There is retraction on the left hemithorax.
- Movement: There is diminished movements on the left hemithorax.

→ By palpation:

- Trachea: There is shift of the trachea to the left side (same side of the lesion).
- TVF: There is increased TVF in the upper lung field supramammary region (cavity) & decreased TVF in the lower lung field (excessive fibrosis).

→ By percussion:

- Note: There is heterogenous dullness on the left side.

→ By Auscultation :

- Breath sound:
 1. Bronchial with bronchophony & whispering pect. in the upper lung field supramammary region (cavity).
 2. Diminished intensity in the lower lung field (excessive fibrosis).
- Additional sounds: Inspiratory medium-sized crepitations in the left side (cavity & fibrosis).

? Why compensated/ not Complicated ?? → No Cor Pulmonale or R.F

► No Cor Pulmonale ::

- From history : no systemic congestion
- Examination: No congested neck veins, no L.L edema, no tender hepatomegaly (in abdominal exam).

► No R.F ::

- History: No cyanosis.
- Clinically: No tremors, cyanosis or disturbed consciousness.
- Lab. : ABG.(the most important as RF is a lab. Diagnosis).

- ? **How would you proceed to manage this case?**
- **Investigation**
 - **For etiology:** for T.B i.e tuberculin test, ESR, CBC "for activity of T.B"
 - **For Functional Diagnosis:** ABG, ECG
 - **For main diagnosis:** CXR, CT, bronchoscope, Pulmonary Function Test (to evaluate for possibility of surgical interference).
 - **Treatment**
 - **Medical:** anti T.B (only if there is evidence of activity), symptomatic ttt.
 - **Surgical:** Resection (in failure of medical ttt or severe recurrent hemoptysis and it must be localized with fair pulmonary functions, otherwise surgery will be contraindicated).
- ? **What organisms tend to cause lung cavitation?**
- Staphylococcus aureus
 - Klebsiella pneumonia
 - Mycobacterium tuberculosis
- ? **What is important to examine in the eyes of this patient ?**
- ☐ Jaundice, because some anti-TB drugs are hepato-toxic as INH, Rifampicin and Pyrazinamide.
- ? **What is the effect of INH on the liver?**
- ☐ Hepato-toxic causing Chronic active hepatitis & Cirrhosis.
- ? **Why would you like to look in the conjunctiva & face?**
- ☐ To look for pallor which might be present in patients with pulmonary TB.
- ? **What other chest diseases may present with pallor ?**
- Other infective chest diseases as: pneumonia, lung abscess.
 - Pulmonary embolism.
- ? **Would you expect to find pallor in this patient ?**
- ☐ No, because:
1. TB is old and not active now.
 2. There is evidence of chronic asthmatic bronchitis.
- ? **Why don't you expect to find pallor in chronic asthmatic bronchitis ?**
- Because in such case, there is usually secondary polycythemia due to the presence of chronic hypoxia which will stimulate the release of Erythropoietin to induce polycythemia.
 - OR: in severe cases of hypoxia, there will be cyanosis.
- ? **What is important to examine in the neck of this patient ?**
- Cervical LN for: TB lymphadenopathy for the possibility of recurrence.
 - Trachea for: position.
 - Neck veins for: congestion in case of development of RVF.
- ? **What investigations do you think were done to the patient when the doctors suspected TB ??**
- Enumerate the investigations of pulmonary TB.
- ? **What do you think Tuberculin test will be in this patient now ?**
- Positive because he got TB before (old TB).
- ? **What makes Tuberculin test positive ?**
- Infection with TB (whether active or old).
 - Vaccination with BCG .
- ? **What do you expect to find in the CXR of this patient on the left side ?**
- Heterogenous opacity with reticulations.
 - The mediastinum moves towards the fibrosis (tracheal shift).
 - The diaphragm moves towards the fibrosis (tenting of the diaphragm).

Bronchiectasis

❖ Personal history...

❖ **Complaint** : Shortness of breath 3 days duration.

❖ HPI:

- The condition started 20 years ago by gradual onset and progressive course of **productive cough**. The sputum is **excessive** in amount, **2 cups** per day, **purulent** in character, **greenish** in colour, increased by **leaning forward**. The patient couldn't detect its odour due to **associated sinusitis**. The patient sought medical advice, investigated by chest x-ray, sputum analysis and tuberculin test which was negative, treated by antibiotics, bronchodilator and expectorant and the condition improved.
- Nine years later, When the patient was 17 years old, he worked as a **welder** and the condition became worse. One year after working as a welder, the patient suffered from gradual onset and progressive course of **exertional dyspnea**. The patient experienced dyspnea on climbing 1st floor with no orthopnea or PND associated with **continuous wheeze**. The patient sought medical advice and admitted in Kasr El-Ainy Hospital, investigated by chest x-ray, sputum analysis and CT chest and treated by inhaler and mucolytics and was advised to stop working as a welder.
- The patient was quite well till 3 days ago when he re-experienced **exertional dyspnea** on climbing 1st floor with no orthopnea on PND.
- **No symptoms suggesting malabsorption, DM, intestinal obstruction and no history of jaundice** (Ask for manifestations of cystic fibrosis).
- **No Haemoptysis**.
- **No pressure symptoms**.
- **No chest pain, cyanosis or systemic congestion**.
- **No Symptoms suggesting other system affection**.

❖ Past history:

- No History of T.B.
- No History of DM, HPN.
- No History of admission to chest sanatorium.
- No history of drugs or operations.

❖ Family history:

- There is similar condition in his family (He has a brother with the same manifestations).
- No consanguinity.
- No common disease in family.

I. General examination :

- The patient is fully **conscious**, well oriented for time, place and person. Average mood and memory. The patient is co-operative with average intelligence.
- **Temperature**: 37° c.
- **BL Pressure**: 130/80.
- **Resp. Rate**: 16/minute, regular, average depth, abdomino-thoracic.
- **Pulse**: Regular, 60 beat/minute, average volume, no special character, equal on both sides, intact peripheral pulsation, vessel wall is not felt.
- **The patient** looks well, average built, **no cyanosis**, pallor or jaundice. He is lying free flat comfortable in bed.
- **Head & Neck**: no puffiness in eyelids, no sub-conjunctival hemorrhage, no working ala nasi or pursing of lips. Neck veins are pulsating **not congested**.
- **Upper limb**: generalized **toxic clubbing** passing from parrot peak to drum stick.
- **Lower Limb**: no L.L edema.

2. Local examination:

A. Inspection:

- **Chest Wall:** no scars, no dilated veins, no pigmentation.
- **Resp. Movement:**
 - **Rate:** 16/minute, regular, average depth, abdomino-thoracic.
 - **Expansion:** bilateral limitation in chest expansion.
 - **Signs of action of accessory Muscles of respiration:** there is suction of supraclavicular fossa, inspiratory indrawing of lower intercostals spaces with no visible contraction of sternomastoid or elevation of thoracic cage (Signs of action of accessory muscles of inspiration). The patient is not pursing his lips or grasping a chair (Signs of action of accessory muscles of expiration). No hoover's sign or tracheal tug (Signs of low flat diaphragm).
- **Shape of the chest:** symmetrical, Barrel shaped chest. (AP diameter $\geq$ Tr. Diameter, raised shoulder, obtuse subcostal angle).
- **Mediastinum:** Central trachea, No trail's sign, No tracheal Tug with **Absent Apex**.
- **Pulsations:** Apex is not visible. There is visible epigastric pulsations(probably aortic).

B. Palpation:

- **Bilateral limitation** in chest expansion.
- **Central Trachea** with decreased tracheal length but no tracheal tug.
- **Absent Apex**.
- **TVF:** equal on both sides.
- **No palpable Rhonci** or rub.
- **No chest wall tenderness**.

C. Percussion:

- **Hepatic dullness** at Rt. 6th Space MCL with normal hepatic span (=14cm) indicating **encroachment on hepatic dullness**.
- **Heart:** No dullness to the right border of sternum, both aortic and pulmonary areas are resonant, preserved waist of the heart, no dullness outside the apex, lower end of the sternum is impaired note with **resonant bare area of the heart** (cardiac dullness is encroached upon by hyper-inflated lung).
- **Lung:** generalized hyper-resonance with encroachment on hepatic and cardiac dullness:
 - **Front:** Resonant clavicles, infra-clavicular areas, dullness is detected at 7th space Rt. and Lt. MCL (You may find basal dullness in these cases).
 - **Lateral:** Dullness at 9th space Rt. And Lt. MAL (You may find basal dullness in these cases).
 - **Back:** Dullness at 11th space Rt. And Lt. SL (You may find basal dullness in these cases).
 - **Resonant Bare area**, Resonant **Kronig's** isthmus.
 - **By Tidal Percussion** → Diaphragm is freely mobile.
 - **No Shifting dullness**.

D. Auscultation :

- Diminished vesicular breath sound with prolonged expiration and diminished V.R. Associated with bilateral diffuse inspiratory coarse consonating **crepitations** changeable with cough. (Scattered expiratory polyphonic rhonchi, specially in upper lung zones, can be auscultated and patchy bronchial breath or areas of diminished air entry can be auscultated in these cases).

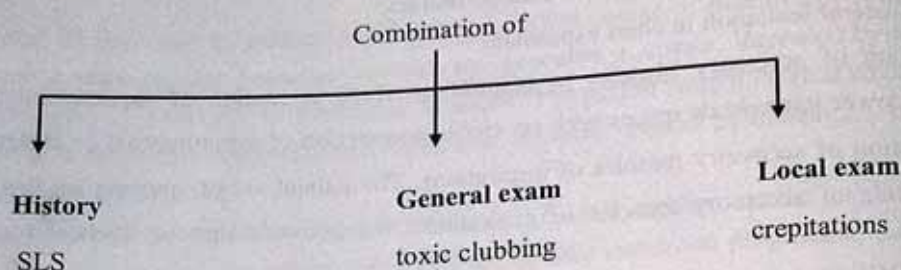
3. Other systems examination...

- **Exposed liver** with no tenderness.

? What is your diagnosis?

- A Case suppurative lung syndrome most probably congenital bronchiectasis associated with sinusitis with signs of hyperinflation in upper lung zones, The patient is compensated not complicated.

? Why Bronchiectasis ??



? Why congenital ?

- From history :: Onset at 8 years old, associated sinusitis (immotile cilia syndrome) and +ve family history.

? Why compensated/ not Complicated ? → No Cor Pulmonale or R.F

► No Cor Pulmonale ::

- From history: No systemic congestion.
- Examination: No congested neck veins, no L.L edema, no tender hepatomegaly (in abdominal exam).

► No R.F ::

- **History:** No cyanosis.
- **Clinically:** No tremors, cyanosis or disturbed consciousness.
- **Lab. :** ABG. (The most important as RF is a lab. Diagnosis).

? How would you proceed to manage this case?

→ Investigation

- **For etiology:** Na in sweat, detection of gene defect (CFTR), measuring cilia motility (nasal biopsy) & (investigations for malabsorption, D.M, intestinal obstruction, obstructive jaundice "cystic fibrosis" should be asked in this patient).
- **For Functional Diagnosis:** ABG, ECG.
- **For main diagnosis:** Sputum analysis, CXR, bronchogram, CT with high resolution, bronchoscopy, Pulmonary Function Tests (for associated COPD and to evaluate for possibility of surgical interference).

→ Treatment

- **Medical:** antibiotics, mucolytics, expectorant, bronchodilator.
- **Drainage:** Postural (Best), bronchoscope, external.
- **Surgical:** Lobectomy, segmentectomy (in failure of medical ttt or severe recurrent hemoptysis and it must be localized with fair pulmonary functions, otherwise, surgery is contraindicated).

? What are the causes of thoracotomy scar?

- Lung malignancy
- Bronchiectasis associated with uncontrolled or recurrent hemoptysis
- Tuberculosis (common in the past)

? What is the most important investigation for this patient ?

- CT chest: (the most important investigation)
- It provides excellent visualization of the bronchiectatic airways.
- It localizes the site & extent of bronchiectasis before surgery.

? What are the other causes of coarse crepitations ?

- Acute pulmonary oedema, lung abscess, late stage of pneumonia (stage of resolution).

? What are the other causes of bilateral basal crepitations ?

- Fine bilateral basal crepitations in case of LVF (pulmonary congestion).

? What is the DD of this case ?

A. Causes of suppurative syndrome; which is defined as:

❖ Cough with expectoration of excessive purulent foetid sputum, usually related to posture :

1. Lung abscess:

- Acute onset , no periodicity.
- Expectoration increases on lying on the healthy side.
- Signs are localized, & Chest X-ray reveals a cavity.

2. Bronchiectasis:

- Gradual onset, periodicity (*more in winter , more in the morning*).
- Expectoration increases on leaning forwards.
- Signs are bilateral & basal, & Chest X-ray reveals honey-combing.

3. Infected cystic lung:

- Signs of bronchiectasis since birth or in early childhood.
- Chest X-ray reveals soap-bubble appearance.

4. Empyema with broncho-pleural fistula:

- Signs of pyopneumothorax.

B. Causes of hemoptysis.

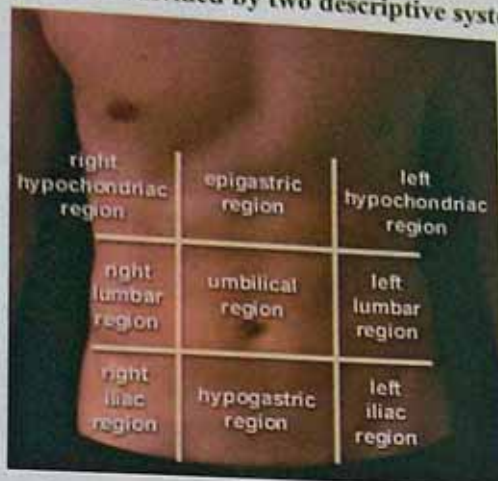
Your Notes

ABDOMEN

❖ Compartments of the abdomen :

- For purpose of description, the abdomen is divided by two descriptive systems into :

9 areas by:



2 Horizontal lines:

- Upper one :
 - Transpyloric plane; midway between suprasternal notch & symphysis pubis (L1) **OR**
 - Subcostal plane (L3): a transverse plane passing through the inferior limits of the costal margin
- Lower one :
 - Supracristal plane: transverse plane passing through the summits of the iliac crests; (L4) **OR**
 - Intertubercular plane: transverse plane passing through the iliac tubercles (L5)

2 Vertical lines :

- Midclavicular line = {mid inguinal line (mid way between symphysis pubis & ASIS)}

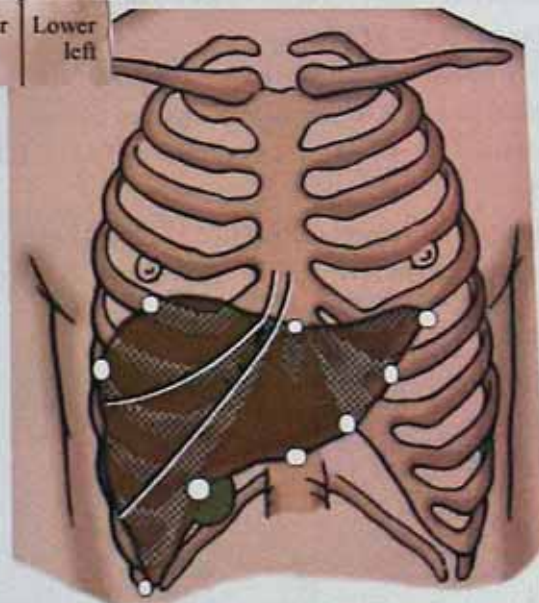
4 areas by :

- Vertical & horizontal line crossing umbilicus



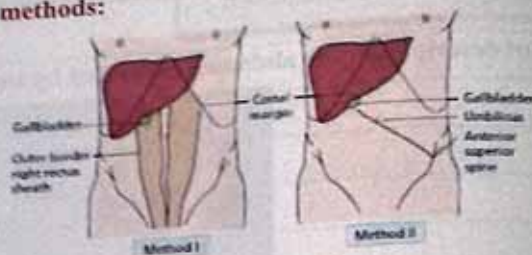
❖ Surface anatomy of the liver:

- Site : Right hypochondrium & epigastric regions
- Upper border : a line connecting :
 - Apex of the heart i.e. left MCL 5th intercostal space
 - A point at the xiphisternum
 - 5th rib in the right MCL
 - 7th rib in the right MAL
 - 9th rib in the right scapular line
- Lateral border :
 - From 7th to 11th rib in right mid axillary line
- Lower border: a line connecting :
 - Tip of right 9th costal cartilage
 - Midway between xiphisternum & umbilicus
 - Tip of left 8th costal cartilage
 - 6th rib in left Mid Clavicular Line
- Normal liver span :
 - Distance between upper & lower border of liver
 - Middle line : 4-8 cm
 - Right Mid Clavicular Line: 8-16 cm



❖ Surface anatomy of the gall bladder :

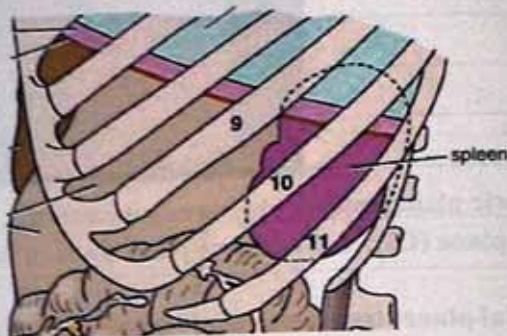
- Normal gall bladder has a fundus, body & neck.
- Surface anatomy of the fundus gall bladder by 2 methods:



- At intersection of lateral border of rectus abdominis with right costal margin
- Intersection of a line {Gray-Turner line} extending from left ASIS to umbilicus with right costal margin at tip of 9th costal cartilage

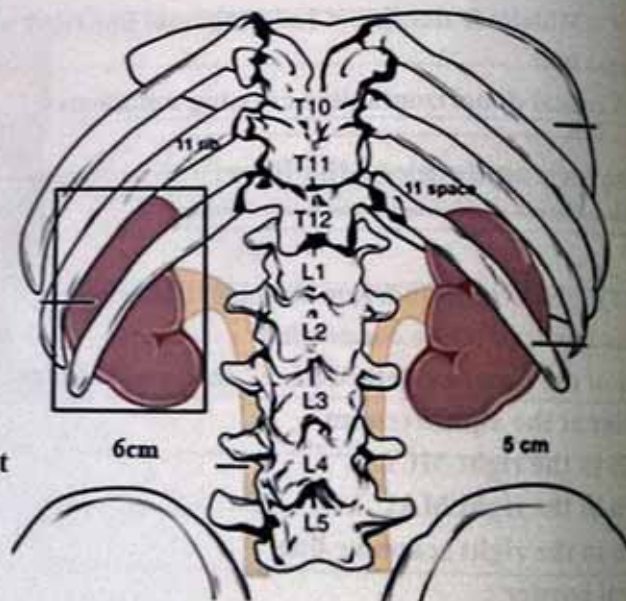
❖ Surface anatomy of the spleen :

- Site : Left hypochondrium
- Lies on the 9, 10 & 11th ribs posteriorly with its long axis in line with 10th rib
- Laterally:** doesn't exceed mid-axillary line
- Medially:** scapular line



❖ Surface anatomy of the kidney :

- Right Kidney is 1 cm Lower than Left Kidney due to the presence of liver
- Posteriorly : bounded by Morris's parallelogram:
 - 2 vertical lines : 3 & 9 cm from median plane
 - 2 horizontal lines : at level of spines of T12 & L3
- Anteriorly:
 - Upper end : 11th space (Right), 11th Rib (Left)
 - Lower end : 5 cm (Rt) & 6 cm (Lt) above iliac crest



Abdominal cases :

1. Bilharziasis: pure Bilharzial fibrosis
2. Hepatitis : Post hepatic cirrhosis
3. Mixed Bilharziasis & Hepatitis cirrhosis
4. Malignancy

ABDOMINAL HISTORY

A. Personal History

1. Name: الإسم ثلاثي	2. Age: ○ Young : hemolytic anemia ○ Old : cancer head of pancreas	3. Sex : ○ Females : cholecystitis ○ Males : cancer head of pancreas
4. Occupation: ○ Duodenal ulcer in stressful occupation ○ Bilharziasis is commoner in farmer	5. Marriage: sterility in liver cirrhosis & Bilharziasis 6. Menstrual history if female.	7. Residence & address: endemic area for Bilharziasis & parasitic infestation • History of travelling abroad
8. Habits of medical importance: ○ Cigarette smoking : for peptic ulcer ○ Alcohol : alcoholic liver cirrhosis - Related to amount, duration e.g. 10L/day for 15 years		9. Handedness.

B. Complaint + duration: إي اللي جابك المستشفى ؟

❖ Common complaints :

▪ vomiting of blood	▪ bloody stool	▪ yellowish discoloration of skin & eye	▪ Abdominal distention
▪ Pain	▪ diarrhea	▪ constipation	▪ Swelling

C. History of present illness = HPI المرض بدأ معاك إزاي . آخر مرة كنت كويس فيها إمتى ؟

1. Pain	2. Swellings: Ascites , Abdominal mass, Lower limb edema, Lymph node
3. Upper GIT symptoms	4. Lower GIT symptoms
5. Hepato-biliary "jaundice"	6. Constitutional symptoms
7. Genitourinary symptoms	8. Uremic symptoms
9. Investigations & treatment received	10. Symptoms of other systems

❖ N.B.:

تكتب في ال complaint بكلام العيان	تكتب في H/O of present illness عادي
الباقى	

Analysis of symptoms mentioned here is the specific data; to be added on the general analysis

1. Pain: ؟ عندك ألم في بطنك ؟

❖ Pain:

❖ Analysis :

- **OCD**, ↑, ↓, Associated symptoms
- **Character**: e.g. colicky مغصى , throbbing نقر , burning حرقان , dull aching ثقل , dragging بطوة او سحب , stitching شكشكة ,... etc.
- **Site** الوجة ده فين
- **Radiation** هل يبسمع في حته تاني
- **Relation to** هل له علاقة ب (meal, respiration, exertion, postural):
- Gastric ulcer increases pain during eating
- Duodenal ulcer increases pain during fasting

2. Swellings: **AALL**

❖ Analysis :

- **OCD**, ↑, ↓, site, size, history of trauma, painful or not;
- **Swellings could be** : e.g. Organomegaly في كلكوعة في بطنك ؟
- A. **Abdominal masses** ؟
- ↳ **Localized Swelling** : بطنك ورمت او نفخت ؟
- B. **Ascites** ؟
- ↳ **Abdominal distension** : بطنك ورمت او نفخت ؟
- **History of tapping** : amount, aspect (clear or turbid) , colour, complications e.g. fever, infection, wound leakage and encephalopathy & recurrence or not.
- C. **Lower limb edema** ؟
- ↳ **Lower limb swelling** : رجلك ورمت او نفخت ؟
- **Value**: liver cell failure , bilharzial cor-pulmonale & nephrotic syndrome : for Analysis (see cardiology)
- D. **Other swellings** في أي كلاكع تاني عندك ؟ e.g. Lymph node
- continue lymphadenopathy sheet

3. Upper GIT symptoms:

- **Appetite** ؟ شهيتك على الأكل اتغيرت ؟
- Anorexia ؟ نفسك مسدودة ؟
- Polyphagia ؟ نفسك مفتوحة ويتاكل على طول ؟
- **Nausea** ؟ نفسك غمة عليك ؟
- **Vomiting** ؟ analysis: حصلك ترجيع ؟
- Spontaneous or induced
- Preceded by nausea or not
- Number of attacks
- Time of occurrence
- Odour & Content
- **Dysphagia** (difficulty in swallowing) , analysis: هل هناك صعوبة في البلع ؟ للسوائل ولا الأكل ؟
- **OCD**, ↑, ↓, Associated symptoms
- Complete (absolute) or partial
- To fluids (achalasia) or to solid (organic)
- Painful (odynophagia) or not
- **Dyspepsia** ؟ الأكل "اللقمة" يتتعبك ؟
- Eructation ؟ يتتكرع كثير ؟
- Water brush ؟ بتحس باللعباب مالي فمك ؟
- **Hiccough** ؟ بيجيلك ز غطة كثير ؟
- **Heartburn** ؟ هل هناك حمو على فم المعدة ؟
- **Hematemesis** ؟ رجعت دم قبل كده ؟

4. Lower GIT symptoms:

- **Flatulence** ؟ بطنك انتفخت ؟ وفيه غازات عندك كثير ؟
- **Diarrhea** ؟ عندك امساك ؟
- **Tenesmus** ؟ هل فيه تعنية او ثقل مع البراز ؟
- **Colour of stool** : لون البراز عامل ازاى ؟
- **Melena** : هل هناك براز اسود زي الزيت وريحته وحشة ؟
- ↳ **Passage of soft black tarry offensive stool**
- **Hematochezia : bleeding per rectum** : هل في دم احمر مع البراز ؟
- ↳ **Bloody stool**

❖ Hematemesis, (portal hypertension), analysis :

❖ Vomiting of blood:

• **OCD**, ↑, ↓, then analysis **ABCD**:

A. Attack : number, date, before the attack..., during the attack..., after the attack ...,

B. Blood : amount, content, color

How can you differentiate between hemoptysis & hematemesis ?

	❖ Hemoptysis	❖ Hematemesis
▪ Before attack:	Cough	Nausea, vomiting
▪ During attack:		
○ Symptoms	Coughing blood	Vomiting blood
○ Colour	Bright red blood	Dark; Coffee-ground (hematine) لون البين المحروق
○ Content	Mixed with air (frothy)	Mixed with food
○ Chemical reaction (pH)	Alkaline	Acidic
▪ After attack	Blood tinged sputum for a while	Melena and constipation
▪ Examination	Chest , CVS signs	GIT signs

C. Circulatory compensation : hospital admission, blood transfusion, treated by (tamponade خرطوم ببلون , endoscopic injection حقن بالمنظار , surgery)

D. Dysfunction : Disturbed conscious level, bleeding from other orifices

... years ago, the patient had {number of attacks....} of Hematemesis of acute onset, {...} course, the attack was in {Date e.g. The fourth of April, nineteen ninety three. (Apr. 1993)} ...was preceded few days before by Nausea & vomiting of {Amount e.g. small or moderate or large amount} of blood, dark red in colour, containing food particles, {±} associated with loss of consciousness, then followed by melena for a ... days, the patient sought medical advice, he was admitted to ... hospital, investigated by ..., received treatment in the form of

5. Hepato-biliary "jaundice" :

❖ Jaundice; analysis :

➤ Yellowish discoloration of skin & eye

1. OCD
2. Anorexia, nausea, vomiting,
3. Bleeding tendency
4. Pruritus ؟ هل فيه هرش ؟
5. Pain (in Right Hypochondrium)
6. Colour (eye, urine, stool)

لون عينك بقى اصفر ؟
ولون البول والبراز اتغير ؟

7. Cachexia & Drugs use
8. Fever, Weight loss & Fatty dyspepsia

7. Genitourinary symptoms :

- ♂ **Genital** : infertility ؟ خلقت قبل كده ؟
○ Male: impotence, libido (↑, ↓) الانتصاب الصباحي ؟
كويس ؟
○ Female: breast atrophy, & menstrual disturbance ؟
في لخبطة في الدورة ؟
- ♂ **Urinary** :
في مشاكل في البول عندك ؟ كمية البول اتغيرت ؟
في دم في البول ؟ في ألم عند التبول ؟ هل بتدخل الحمام كثير ؟
○ Amount of urine
○ Pain , Passage of stones
○ Color (hematuria)
○ Dysuria , urgency, frequency (normal 5-6/day, 0-1/ night)

6. Constitutional symptoms :

في ارتفاع في درجة الحرارة ؟ فيه صداع ؟ في فقدان للشهية ؟
في تعب وهمدان في جسمك ؟

1. In LCF & malignancy & TB peritonitis ask about:
 - Fever, headache, anorexia , malaise, fatigue, sweating & loss of weight
 - Bleeding tendency
2. In splenomegaly ask about:
 - Other swellings e.g. Lymph node all over the body
 - Bony pains : in leukemia
 - Bleeding tendencies in skin & from orifices :

بتنزف من اسنانك او بتجيب دم من انفك ؟
بتتطلعك بقع في جسمك مع اي خبطة صغيرة ؟ في نزيف مع البول او البراز ؟

8. Uremic symptoms

- Anorexia , nausea, vomiting
- Bad odor of breath "halitosis" راحة فم متغيرة ؟
- HicCough
- Disturbed Conscious Level (DCL), headache, lack of concentration

10. Symptoms of other systems :

- هل عندك مشاكل تالية في جسمك
وتأخذ كل سيستم وتسال العين على الاعراض الكبيرة اللي فيه بسرة اوي

D. Past history as usual but focus on the followings:

A. Diseases:

○ Intestinal parasites :

→ **Contact with water canals for Bilharziasis**

← جالك بلهارسيا قبل كده ؟ نزلت التربة قبل كده وجالك دم في البول او البراز ؟ لو نعم كمل اسئلة

← الحكاية ديه من امتي ؟ اخدت علاج اي ؟ جرعتة اداي ؟ حللوك بعدها وقالوك خفيت ولا لا ؟ نزلت التربة بعد كده ؟

○ Common disease : DM, TB & hypertension

○ Viral hepatitis : blood transfusion , infection ؟ نقلت دم ، سخذت وعينك اصفرت وبولك اغمق وروحت الحميات ؟

○ Fever : typhoid & brucellosis

B. Operations & blood transfusion : hepatitis especially HCV

→ History of recurrent blood transfusion (not regular in cirrhosis , repeated regular in hemolytic anemia)

C. Drugs: hepatotoxic drugs : see later

E. Family history as usual & asked for familial polyposis & hemolytic anemia.

DISCUSSION ON HISTORY

❖ What are the types of abdominal pain?

⊗ Types of Abdominal pain:

A. Visceral	B. Parietal "Somatic"	C. Referred
- Felt at the site of the primary stimulus. - Vague, Not localized & difficult to describe - Subtypes : - Colic : from hollow organs (obstruction) - Dull ache : from hollow or solid organ (distention)	- Irritation of the parietal peritoneum - Sharp, localized & easier to describe	- Pain felt at a site other than stimulated but in area supplied by the same neural segment - E.g.: cholecystitis → right shoulder

❖ What are the abdominal causes of chest pain ? = referred pain

Amebic liver Abscess	Subphrenic Abscess	Peritonitis	Hiatus hernia
Peptic ulcer	Cholecystitis	GORD	

❖ What are the non-alimentary (non-abdominal) causes of abdominal pain?

Cardiac : myocardial infarction	Diabetic ketoacidosis	Herpes zoster
Chest : acute pleurisy & pneumonia	Dissecting aortic aneurysm	Torsion testis & ovary
Cord compression (thoracic segment)		

❖ What are the medical causes of acute abdominal pain?

Addisonian crisis	Porphyria	Severe hypercalcemia
Hemolytic crisis	Sickle cell anemia	Cardiac : myocardial infarction
Chest : acute pleurisy & pneumonia	Diabetic ketoacidosis	Systemic lupus erythematosus
Rheumatic fever (non-infective peritonitis)	Familial mediterranean fever	Food poisoning & Uremia

❖ What are the types of splenic pain?

Dull ache : heaviness due to stretch of capsule	Dragging : stretch of ligaments by weight of enlarged spleen	- Throbbing : abscess - Stabbing : due to infarction - Stitching : due to perisplenitis
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❖ What is the definition of :

A. Anorexia	B. Polyphagia	C. Parorexia
Loss of appetite	Increased appetite	Perverted appetite (Picca)
What are the causes of :		
Anorexia?	Polyphagia with progressive loss of weight?	Pica?
- Psychogenic : anorexia nervosa & depression - GIT : atrophic gastritis , cancer, viral hepatitis - Debilitating diseases : TB, liver Failure, renal Failure, malignancy	- Mal-absorption syndrome - Parasitic infestations - Thyrotoxicosis - DM	- Ankylostoma Anemia - Pregnancy, Psychogenic - Ca deficiency

❖ What are the causes of Halitosis? ; Bad mouth odour :

Oral cavity : caries, pyorrhea, tonsillitis, sinusitis	Esophagus : diverticulum & cancer	Intestine : malabsorption, chronic intestinal obstruction
Suppurative lung syndromes	Liver cell failure "fetor hepaticus"	Chronic renal failure "ammoniacal odour"

❖ What are the causes of Hiccough?

- Definition :** Abnormal spasmodic contraction of diaphragm due to irritation of phrenic nerve or diaphragm

• Causes :

- Idiopathic : commonest.
- Chest: pericarditis, empyema & mediastinal syndrome
- Diaphragm irritation : subphrenic, liver or splenic abscess
- Renal failure "uremia" : common known cause

❖ What are the causes of jaundice after blood transfusion?

- Incompatible blood transfusion
- Hepatitis
- Liver cell failure

❖ What is the color of blood in hematemesis ?

- Dark red due to acid hematin which indicates gastric origin

❖ Could it be bright red in colour ? yes, in the following conditions:

- Massive rapid severe bleeding
- Esophageal varices
- Achlorhydric stomach

❖ What are the commonest causes of hematemesis ?

- Peptic ulcer
- Esophageal varices
- Cancer stomach & drug erosion

❖ What is the value of hematemesis ?

- The patient is vascularly decompensated
- Change lines of treatment

❖ What are the causes of loss of consciousness after hematemesis ?

- Vasovagal shock
- Hypovolemia
- Hepatic encephalopathy

❖ What is the difference between melena & hematochezia ?

	Melena	Bleeding per rectum (hematochezia)
• Definition :	○ Passage of digested blood with stools	○ Passage of fresh blood with stools
• Site of the lesion	○ Hematemesis or melena usually indicate a gastrointestinal bleeding from a lesion above ileocecal valve : A. Above ligament of Trietz : upper GIT causes ; esophagus , stomach & duodenum B. Below the ligment of Trietz : lower GIT causes : jejunum & ileum	○ Hematochezia usually indicates gastrointestinal bleeding below ileocecal valve i.e. lower gastrointestinal bleeding i.e. colon & anorectum
• N.B.:	• Right side colonic lesions are usually associated with melena or bleeding per rectum while left side colonic lesions are associated with bleeding per rectum	
• Amount	○ Amount : > 50 – 100 ml blood if less than this it will be occult blood in stool	
• Duration	○ > 8 hours; time needed for blood to be broken down within the intestinal lumen	○ < 8 hours ; rapid transit time due to massive severe brisk bleeding
• Character	○ Black tarry, glistening , soft offensive	○ Bright red in color
• Differential diagnosis	○ Iron, charcoal , bismuth	○ Diverticulosis, angiodysplasia, infectious colitis

❖ What is meant by spurious melena?

- Due to ingestion of blood of epistaxis or haemoptysis

❖ How does the patient pay attention to melena?

- Blood in intestine → diarrhea → attention

❖ How could you differentiate between melena & iron therapy as a causes of dark stool?

	Melena	Iron therapy
	-ve	+ve
• History of drug intake	Diarrhea "blood is irritant to GIT"	Constipation
• History of bowl habits		Hard
• Stool examination : consistency	Soft	Usual odour of stool
• Stool examination : Odour	Offensive	
• Dilute the stool by water :	Red	Grayish

❖ How to detect occult blood in stool ?

- 1) Guaiac test : stool + ether + guaiac reagent → blue color in positive cases
- 2) Benzidine test : less reliable as it gives positive results with iron therapy
- 3) Isotopic method : RBCs labeled with Cr 51

❖ What are the causes of jaundice & fever ?

A. Hemolytic jaundice + fever
○ Hemolytic crisis ○ Pulmonary embolism ○ Incompatible blood transfusion ○ Malaria

B. Obstructive jaundice + fever
▪ Charcots triad : fever & rigors + Jaundice + biliary colic (pain) ○ Malignancy

C. Hepatocellular jaundice + fever
○ Viral hepatitis (preicteric phase) ○ Liver cell failure ○ Hepatoma

❖ What are the hepato-toxic drugs you know?

A. Dose dependent e.g. Acetaminophen (Paracetamol)

- It's safe in usual doses
- Indication : analgesic antipyretic, used when aspirin is contraindicated as in:

▪ Peptic ulcer	▪ Bronchial asthma	▪ Pregnancy	▪ Bleeding tendency	▪ Aspirin allergy
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• Toxic dose : 15 gm (30 قرص)

• Antidote: N-acetyl cysteine

B. Dose Independent :

▪ Acute hepatitis	INH	Alcohol	Halothane	Paracetamol
▪ Chronic hepatitis	INH	Alcohol	Methyldopa	DDT
▪ Cirrhosis	INH	Alcohol	Methyldopa	Methotrexate
▪ Cholestasis	Androgen	Estrogen	Chlorpromazine	
▪ Tumors	Androgen	Estrogen		
▪ Fatty liver	Alcohol	Steroids	Valproic acid	Tetracycline

❖ What are the definition & causes of heart burn = pyrosis = cardialgia?

- Definition : Vague retrosternal burning sensation due to acid regurgitation from stomach
- Aggravated by recumbence, leaning forward & meal
- Relieved by antacids

• Causes :

- Functional : stress
- Organic : GORD, hiatus hernia, peptic ulcer

❖ How can you differentiate between heart burn & ischemic heart?

	Heart burn	Ischemic heart
Aggravated by	Recumbence, leaning forward & meal	Exercise
Relieved by	Antacids	Rest & nitrates

❖ How can you differentiate between esophageal spam & ischemic heart?

	Esophageal spam	Ischemic heart
ECG changes	Present	Present
Nitrates	Relief	Relief
Endoscopy	The only diagnostic way to differentiate between both	

❖ What are the definition & causes of :

A. Diarrhea : Frequent bowel motions (> 4 / day) or loose stools or both

- Causes : causes of dysentery

B. Tenesmus: تَغْنِيَة

- Frequent painful desire to defecate + sense of incomplete bowel evacuation
- Causes : rectal lesion (inflammation or mass)

C. Dysentery: diarrhea + tenesmus + blood + mucus in the stool إسهال مع دم ومخاط أبيض + تَغْنِيَة

• Causes are :

▪ Infective:	○ Bacterial (bacillary)	○ Protozoal (ameba, giardia, malaria)	○ Metazoal (bilharzia)
▪ Metabolic: uremic	▪ Toxic: mercury poisoning	▪ Local: diverticulosis, ulcerative colitis, cancer col	

D. Constipation : infrequent evacuation of bowel (> 48 hr.) + passage of hard small stools

- Causes: lack of fiber & fluid intake, myxedema, TB peritonitis & typhoid fever.

❖ What are the medical causes of itching (pruritus)?

• Obstructive jaundice	• Primary biliary cirrhosis	• Diabetes mellitus
• Bilharziasis	• Drug hypersensitivity	• Chronic renal failure
• Hemolytic anemia	• Polycythemia	• Lymphoma & leukemia

❖ What are the significance of time, content & odour of vomiting ?

A. Time :	B. Contents :
○ Early morning : CNS ○ During meals : esophageal obstruction ○ 1/2 hr. After meals : gastric ulcer ○ 2-3 hr. After meals : duodenal ulcer ○ > 8 hr. After meals : pyloric obstruction	○ Mucus : chronic Gastritis & malignancy ○ Fecal matter : intestinal obstruction ○ Bile : regurgitation of duodenal contents ○ Parasites : ascariis ○ Blood : peptic ulcer & cancer stomach ○ Stone : cholecystogastric or duodenal fistula
C. Odour :	
○ Foul : pyloric obstruction & cancer stomach ○ Fecal : intestinal & gastrocolic fistula	

❖ What are types of pain related to uro-genital system?

• Renal : dull ache in loin referred to hypochondrium	• Ureteric : colicky from loin to groin	• Bladder : suprapubic referred to tip of penis
• Prostatic : bladder pain + perineal pain	• Urethral : burning (scalding) pain during micturition	

❖ What are the micturition disturbances related to uro-genital system?

• Dysuria : pain experienced prior to, during or after micturition (gonorrhea)	• Strangury : in addition to dysuria there is intense desire to urinate every few minutes without relief of pain = vesical tenesmus	• Frequency: ↑ frequency of micturition in cystitis, DM, prostatic enlargement (urine volume may be constant)
• Urgency : strong desire to micturition (cystitis), Incontinence occur if no opportunity to urination	• Precipitancy: severe urgency, Patient can not hold his urine (UMNL)	• Hesitancy : difficult to start micturition (prostate)
• Second micturition : in large bladder diverticulae	• Retention : acute or chronic • Enuresis : involuntary voiding of urine during sleep	• Difficult to maintain act: ○ ↓ by straining : urethral stricture ○ ↑ by straining : prostate
• Stream abnormalities : continuous or interrupted, thick or thin, strong or weak, biforked or one stream		

❖ What are urine abnormalities in volume, colour, content?

A. Volume abnormalities:	B. Colour abnormalities:	C. Abnormal contents :
• Polyuria : volume of urine > 1500 CC / 24h. • Oliguria : volume of urine < 400 CC. / 24h. • Anuria : complete absence of urine > 12-24 hours	• Red : hematuria, hemoglobinuria, food (beet root) & drugs (Rifampicin) • Dark (tea-like) : obstructive & hepatocellular jaundice • Milky or cloudy : pyuria, phosphaturia, chyluria	• Blood (hematuria) : 3 glass test; ○ Initial : urethral ○ Terminal: bladder ○ Total : renal • Shreds of tissues (necroturia) : in bladder cancer • Stones : Number, size, colour & shape • Gas (pneumaturia) : in vesicocolic fistula

❖ What are the definition & causes of :

❖ Salivation disturbances :

⊗ Ptyalism: Increased salivation ريقى سايب	⊗ Xerostomia "aptyalism" : Dry mouth ريقى ناشف
Causes: ▪ Mouth breathers ▪ Sjogren's syndrome ▪ Dehydration, Depression, Drugs : atropine ▪ DM, Uremia	
▪ True : inflammation & tumors of tongue, gums ▪ False: facial palsy	

General Examination Of Abdominal Case

❖ Examination as general examination but stress on the following:

I. Vital signs			
❖ Temperature	❖ Pulse	❖ Blood pressure	❖ Respiratory rate
❖ Low grade fever in LCF ❖ High grade fever in viral hepatitis & spontaneous bacterial peritonitis	❖ Tachycardia & Water hammer pulse in LCF: vasodilator materials ❖ Bradycardia in obstructive jaundice	❖ Decrease diastolic pressure (VD) in advanced LCF	❖ Rhythm: Irregular in hepatic coma ❖ Type: thoracic-abdominal

II. General overview

A. Appearance	B. Built	C. Consciousness	C. Colors	D. Decubitus	E. Expression
▪ Emaciation = Wasting: ▪ Spider man: combination of thin limbs & protuberant abdomen due to ascites due to liver cirrhosis	▪ Underbuilt : - Stunted growth in chronic liver disease since childhood ▪ Overbuilt : - Autoimmune hepatitis treated by cortisone - Cholecystitis	▪ Disturbed in hepatic encephalopathy	❖ Pallor in anemia in LCF ❖ Cyanosis : LCF ❖ Jaundice : refer to general chapter ❖ Hyperpigmented : in hemochromatosis & LCF	▪ Orthopnea in tense ascites	▪ Mongolian facies in chronic hemolytic anemia

❖ What are the mechanisms of central cyanosis & clubbing in LCF?

1. Opening of pulmonary AV shunt due to vasodilator material
2. Porto-pulmonary shunts
3. Basal lung collapse due to tense ascites

III. Systemic overview :

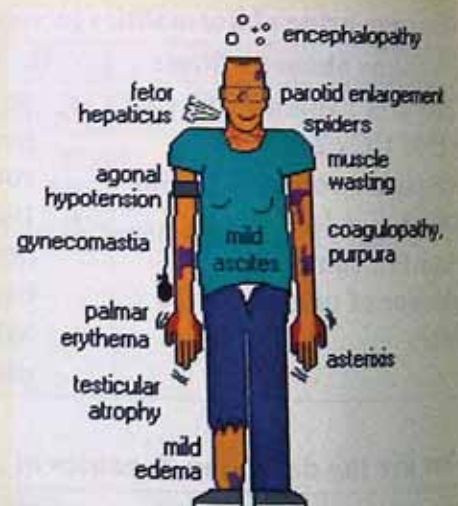
❖ Head & Neck

- Cornea: Kayser-Fleischer rings : in Wilsons disease : greenish brown rings
- Hyperpigmented macules on the lips and oral mucosa (Peutz-Jeghers syndrome)
- Thyroid swelling with Lupoid hepatitis
- LN enlargement in lymphoma & leukemia & TB

❖ **Manifestation of liver cell failure in head & neck :**

- ⊗ **Hepatic encephalopathy** (terminal) due to increase ammonia: **SAD**
 - Sleep disturbances (somnolence & inverted sleep rhythm),
 - Speech disturbances (slurred speech or monotonous speech)
 - **Apathy, Asterix**
 - Disturbed personality & behavior
 - **DCL** with disorientation for time, place & person
- ⊗ **Fine silky hair**
- ⊗ **Jaundice** (hepatocellular): orange yellow sclera, pale stool, dark urine
- ⊗ **Pallor** in conjunctiva, lips; all types of anemia.
- ⊗ **Central cyanosis** in tongue
- ⊗ **Foetor hepaticus** : faecal odour simulating rotten apple due to lack of demethylation of methyl mercaptan
- ⊗ **Skin**: spider naevi, xanthelasma, bleeding tendency
- ⊗ **Endemic parotitis** : bilateral painless enlarged parotid
- ⊗ **Congested neck veins** (cor-pulmonale & massive ascites)

- ❗ If spider naevi disappeared in a hepatic patient ... what are the possibilities?
- ◆ Improvement
 - ◆ Terminal (hypovolemia → hypodynamic circulation)



❖ Manifestation of liver cell failure in limbs (Upper & Lower limbs):

- ⊗ Asterixis : Flapping tremors
- ⊗ Palmar erythema
- ⊗ Palmar or tendon xanthomata
- ⊗ Pallor "Anemia"
- ⊗ Pigmentation in hemochromatosis, primary biliary cirrhosis & extra-hepatic manifestation of HCV.
- ⊗ Paper money skin : numerous small vessels scattered through the skin in random fashion due to excess estrogen
- ⊗ Capillary pulsation : hyperdynamic circulation
- ⊗ Cyanosis
- ⊗ Cyanotic clubbing
- ⊗ Changes in Nail:
 - Koilonychia : spooning in iron deficiency anemia
 - Leukonychia: white opaque in hypoalbuminemia
 - Muehrcke's nails: transverse white bands parallel to lunula in hypoalbuminemia
- ⊗ Dupuytren Contracture in alcoholic liver cirrhosis
- ⊗ Edema : bilateral pitting lower limb edema
- ⊗ Spider naevi
- ⊗ Scratch Marks
- ⊗ Subcutaneous hemorrhage : Bleeding tendency : petechiae, purpura & ecchymosis
- ⊗ Skin & Muscles : Warm hand & Wasting of muscles



❖ What is the mechanism of bleeding tendency in LCF ?

A. Defect in platelets:

- Thrombocytopenia "decreased platelets" due to hypersplenism
- Thrombocytopathy due to diseased platelets due coating by toxins

B. Defect in clotting factors :

- Deficiency of :
- Prothrombin, Fibrinogen
 - Factors : V, VII, IX , X

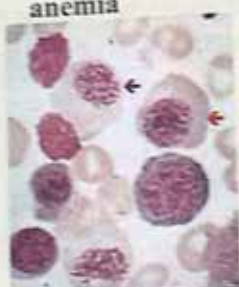
❖ What are the causes of edema of lower limb & ascites in abdominal case?

○ Liver cirrhosis	○ Mal-absorption syndrome	○ Protein losing enteropathy
○ Bilharzial cor pulmonale	○ Malnutrition	○ Nephrotic syndrome

IV. Other systems except that of local exam :

❖ Cardiac examination	❖ Chest examination	❖ Renal examination	❖ Neurological examination
▪ As a cause : ○ Cardiac cirrhosis ○ SBE = splenomegaly ▪ As a result : ○ Bilharziasis ○ Cor- pulmonale	▪ As a cause : ○ Core-pulmonale ▪ As a result : ○ Amoebic liver abscess ○ Hydatid cyst ○ Hepato-pulmonary syndrome ○ Guillian Barre syndrome (extra-hepatic complication of HBV & HCV) ▪ As an association : ○ Alpha one antitrypsin deficiency	▪ As a result : ○ Hepato-renal syndrome ○ Pre-renal failure (acute GIT bleeding) ○ Nephrotic syndrome (extra-hepatic complication of HBV & HCV, schistosoma mansoni)	▪ As a cause: ○ Autoimmune peripheral neuropathy (gastroparesis diabeticorum) ▪ As a result : ○ Hepatic encephalopathy ▪ As association : ○ Hepato-lenticular degeneration "Wilson's disease)

❖ **Manifestation of vitamin deficiencies as findings during general examination:**

❖ Manifestation of vitamin deficiencies		
⊖ Follicular hyperkeratosis (goose like skin)	⊕ Vitamin A (Retinol) ⊖ Xerophthalmia : Bitot's spots : dryness & keratinization of conjunctiva	⊖ Keratomalacia, night blindness
		
⊕ Vitamin D (Cholecalciferol)	⊕ Vitamin E (Tocopherols), ⊕ Vitamin B7 (Biotin)	⊕ Vitamin K (phyloquinone)
⊖ Rickets or osteomalacia (bone pain & pathological fracture)	⊖ Dermatitis	⊖ Hypoprothrombinemia (bleeding tendency)
		
⊕ Vitamin C (ascorbic acid)		⊕ Vitamin B1 (thiamine)
⊖ Scurvy (bleeding gums)	⊕ Folic acid (Vitamin B9) ⊖ Megaloblastic anemia	⊖ Beri - Beri
		
⊕ Vitamin B2 (Riboflavin)		
⊖ Angular stomatitis & cheilitis, Red glazed tongue	⊖ Circumcorneal vascularization	⊖ Pityriasis alba
		
⊕ Vitamin B5 (Pantothenic acid) Peripheral neuritis	⊕ Vitamin B12 (cobalamin)	
	⊖ Megaloblastic anemia ⊖ GIT : glossitis, dyspepsia, diarrhea ⊖ CNS : - Subacute combined degeneration of spinal cord	
⊕ Vitamin B6 (pyridoxine): Dermatitis & peripheral neuritis		
⊕ Nicotinic acid (Niacin) (Vitamin B3) :		
⊖ Pellagra : Pellagic rash : ▪ Site on sun exposed & pressure areas (heels, knees & trochanters) ▪ Description : - Hyperkeratosis (rough skin, scaly desquamated) & Hyperpigmentation ⊖ Red glazed tongue		
		

Local Examination Of Abdominal Case

Rules :

1. Good light
2. Exposure the patient's abdomen from nipples down to knees
3. Position of the patient :
 - Supine position with his arm at sides and knee slightly flexed to relax the abdominal wall (muscles & Scarpa's fascia which is the membranous layer of the superficial fascia of the abdomen)
 - Sitting, if the doctor will examine the back
 - Standing, if the doctor will examine for hernia & genitalia
4. Examine from the patient's right side
5. Make sure that the patient is comfortable in this position

Examination:

I. Inspection	II. Palpation	III. Percussion	IV. Auscultation
❖ Inspection : Anterior + Posterior			
📖 2 general	📖 7 in midline		
▪ Contour of the abdomen ▪ Movement with respiration	▪ Subcostal angle ▪ Divarication of recti ▪ Umbilicus ▪ Epigastric pulsation ▪ Visible peristalsis ▪ Suprapubic hair distribution ▪ External genitalia	📖 8 on sides	
		▪ Breast ▪ Dilated veins ▪ Scars ▪ Scratch marks ▪ Striae ▪ Subcutaneous hemorrhage ▪ Edema of abdominal wall ▪ Hernial orifices	

I. Inspection of the **AB**domen = **A**nterior + **B**ACK

❖ Anterior Aspect : general , midline, sides

❖ 2 General:

1) Contour of the abdomen (transverse & antro-posterior diameter)



⊗ **Technique** : Tangentially من حرق السرير ومرة من الجنب

⊗ **Normally**:

- ☞ Slightly scaphoid from up downwards
- ☞ Preserved waist (convex) from side to side
- ☞ With empty flanks



Normal

⊗ **Abnormally** :

1. Marked Retraction (scaphoid): starvation, dehydration & wasting diseases e.g. malignancy, TB
2. Bulging (distention): localized or generalized :

A. Localized = Visible masses e.g. organ swelling: liver, spleen, ... etc.

❖ **Comment on**:

⊗ **Rising up test & observe the mass** حط ايدك على راس العيان وقوله هم براسك

→ Extra-abdominal mass: Increase in size

→ Intra-abdominal mass: Decrease in size

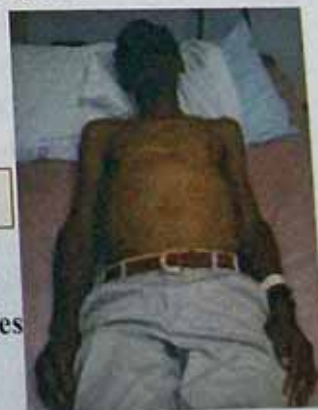
⊗ **Movement with respiration**:

→ Extra-abdominal mass : moves in & out with respiration if related to abdominal muscles

→ Intra-abdominal mass : moves up & down with respiration if related to diaphragm

⊗ **Site , size, shape, surface, , skin & Pulsations**

Localized Bulge →



B. GENERALIZED abdominal distention , Causes :

Ascites one of the signs of LCF

1. Fluid : ascites , ovarian cyst	2. Full bladder	3. Faeces	4. Fat (obesity)
5. Fetus: signs of pregnancy	6. Flatus: hyper-resonance all over the abdomen	7. Fibroma	

❖ How to differentiate between ascites (free fluid) & ovarian cyst (encysted fluid) ?



	Ascites	Ovarian cyst
▪ Distention	▪ Diffuse	▪ Mainly central
▪ Umbilicus	▪ Shifted downward	▪ Shifted upward
▪ Percussion	▪ Central resonance & dull flanks	▪ Central dullness & resonance flanks
▪ Shifting dullness	▪ Bilateral	▪ May be unilateral
▪ By US	▪ Free fluid	▪ Encysted fluid



Ovarian cyst

❖ How to differentiate between ascites & obesity?



Ascites	Obesity
Umbilicus is everted	Umbilicus is tucked in



2) Movement with respiration:

- ❖ Technique : tangentialي العيان يتنفس عادي وبص من الجنب
- ❖ Normally : abdomen moves freely with respiration (bulge during inspiration & retract during expiration)
- ❖ Abnormally :

○ Limited: Tense ascites	○ Absent: Acute peritonitis
○ Diaphragmatic paralysis:	
○ Unilateral : one side moves only (See-saw movement)	
○ Bilateral : paradoxical movement i.e. opposite the normal	

❖ 7 in Midline:

1) Subcostal angle:

- ❖ Technique : thumb test at xiphi-sternal junction
- ❖ Normally : acute to right angle = 90° (60° – 110°):
 - Tall & thin : more acute : 60°
 - Short & obese: obtuse : 110°
- ❖ Abnormally : wide obtuse subcostal angle (180°) , causes:
 - Chronic increased intra-abdominal pressure (upper swellings & ascites)
 - Chronic increased intra-thoracic pressure e.g. COPD



2) Divarication of recti

- ❖ Definition : widening & stretching of linea alba
- ❖ Etiology : chronic increase in intra-abdominal pressure (upper swellings & ascites), (it may be congenital)
- ❖ Types : complete (above & below umbilicus), partial (one side)
- ❖ Differential diagnosis : fatty hernia of linea alba & epigastric hernia
- ❖ Technique :



طابق على رأس العيان وقوله بهم برأسه وبص على ال recti
 لطاينك عمودية على البطن وحاول تدخل أطراف الأصابع بين ال recti
 لم الطبيعي ← متقدش تدخل صوابك
 في Divarication ← تقدر تدخل صوابك وتحرك من جنب لجنب.

3) Umbilicus : 4S + 2D

1. Site :

- ❖ Normally : midway between xiphisternum & symphysis pubis
- ❖ Abnormally :
 - Shifted downwards in Ascites & upper abdominal swellings : hepatic & splenic enlargement
 - Shifted upwards in pelvi-abdominal masses

2. Shape :

- ❖ Normally : inverted (attached to umbilical ligament)
- ❖ Abnormally : everted in increased intra-abdominal pressure (upper swellings & ascites) or in umbilical & paraumbilical hernia

3. Swellings:

- Nodule: endometriosis & Sister – Mary Joseph nodule
- Hernia : expansile impulse on cough



in GIT, Liver, Breast Carcinoma

→ What are other causes of expansile impulse on cough ?

Meningocele, laryngocele, pneumatocele, empyema necessitans, saphina varix & hernia

4. Skin pigmentation:

- Addison's disease & TB peritonitis
- Cullen's sign : bruising around the umbilicus suggests intra-peritoneal bleeding
- Gray Turner's sign : bruising in flanks area, suggests retroperitoneal bleeding

5. Skin lesions:

- Scars & Ulceration
- 6. Dilated veins in caput medusa due to portal hypertension
- 7. Discharge :

- Pus : inflammation, Stool : intestinal fistula, Urine : patent urachus

→ What are the signs of chronic increased intra-abdominal pressure ?

- Wide subcostal angle.
- Divarication of the recti.
- Umbilicus: everted, shifted downwards ± umbilical hernia.



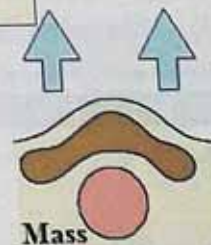
4) Epigastric pulsations: see cardiology

- RV enlargement : in Bilharzial cor pulmonale
- Hepatic pulsation in tricuspid valve disease
- Aorta : normal, aortic regurge, aneurysm or solid mass transmitting aortic pulsation

→ How can you differentiate between aortic pulsation or solid mass transmitting aortic pulsation ?



Aorta



Mass

	Aortic pulsation	Solid mass
▪ Two finger test	Expansile pulsation i.e. pulsates laterally & antro-posterior i.e. in all direction	Transmitted pulsation i.e. pulsates antro-posterior only
▪ Pulsations on Knee- elbow position	Not affected	Disappears

5) Visible peristalsis

A. Causes :

- ❖ Occasional finding in normal thin individuals
- ❖ In pyloric obstruction : slow waves in upper abdomen, moves from left to right
- ❖ In small intestinal obstruction : step ladder pattern around umbilicus
- ❖ Colonic obstruction : Horse shoe pattern

B. Techniques to Stimulate it:

- ❖ Gentle tapping, or massage
- ❖ Cold stimulation of skin (2 drops of ether)
- ❖ Drinking soda water
- C. Confirmed by succussion splash "in auscultation"



6) Supra-pubic hair distribution

❖ Normally :

- Male : triangular with apex towards umbilicus (Diamond shape)
- Female : triangular with upper horizontal line



❖ Abnormally

Feminine distribution "straight instead of triangular" in LCF due to lack of destruction of estrogen

- Lost hair in male & female : hypogonadism

7) External genitalia العيان والف

❖ Importance of genitalia in abdominal case:

- Testicular atrophy by consistency (small & soft) with LCF
- Thick spermatic cord: Bilharzial mass
- Scrotal edema in nephrotic syndrome
- Hydrocele in ascites
- Varicocele intra-abdominal tumour
- Sinus in back of scrotum TB peritonitis

❖ 8 on sides :

1) Breast:

- Atrophy of female breast in LCF



Atrophic Breast

- Gynecomastia:



Gynecomastia

- Definition : hypertrophy of glandular tissues of male breast with prominent nipples
- Characters : firm & tender, $\pm$ bilateral
- Causes of gynecomastia in liver cirrhosis : LCF ($\uparrow$ estrogen) - drugs (spironolactone, digoxin)
- Diagnosis : dick test $\rightarrow$ firm tender disc beneath the nipple

index و ال thumb على جانبي ال breast من تحت خالص وتطلع لفوق لغاية ال nipple

Gynecomastia $\leftarrow$ disc زي ال

no gynecomastia $\leftarrow$ no disc

3) Dilated veins:

❖ Veins which are found on the abdominal wall may be either :

	A. Just Visible veins	B. Abnormally Dilated veins
		
Cause	Tense ascites	Obstruction
Shape	Straight	Tortuous
Diameter	Narrow	Wide
Level	Skin level	Elevated
Direction	Normal	Abnormal

❖ Veins which are confirmed to be dilated may be either :

- $\rightarrow$ Portal veins obstruction: portal hypertension (caput medusa)
- Caput medusa : dilated veins radiating away from the umbilicus & caused by reopening of umbilical vein by the high pressure of portal vein in case of portal hypertension
- $\rightarrow$ Systemic veins obstruction (SVC or IVC)

Caput medusa $\rightarrow$



Differential Diagnosis between them :

Inspection : site	Portal obstruction "caput medusae"	Systemic obstruction
Palpation : direction of blood flow	Center , around umbilicus	Peripheral
Auscultation : venous hum	Away from umbilicus	SVC: from up down
Investigations	Yes: venous hum	IVC: from down up
Infrared photography	Diagnostic	No
Oral glucose test (old test)	Higher concentration	Diagnostic
Oral glucose test : Glucose absorbed from the intestine to the abdominal veins i.e. portal venous blood, which now has higher glucose concentration than that of systemic veins		No difference

Systemic veins obstruction may be either:

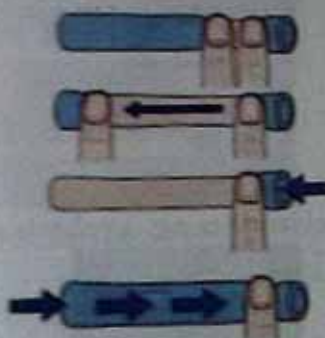
→ SVC obstruction

→ IVC obstruction

Differential Diagnosis between them :

	SVC obstruction	IVC obstruction
		
Direction	Above downwards	Below upwards
Site	Crosses costal margin Upper abdomen & chest	Crosses the inguinal ligament Lower abdomen

Determination of direction of blood flow by: milking technique → rapid refilling



- اختار segment تكون أطول ما يمكن و without tributaries
- فصلها من الدم بال 2 indices
- ارفع ال index اللي فوق وشوف سرعة امتلاء ال vein
- ثم فصلها مرة أخرى
- ارفع ال index اللي تحت وشوف سرعة امتلاء ال vein
- في المرتين ال vein هيتملئ ولكن الأسرع فيهم هو اللي هيحدد اتجاه الدم

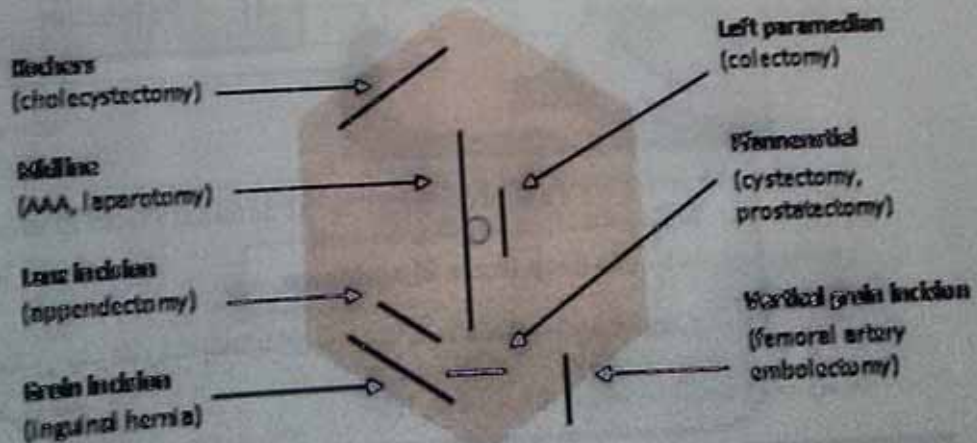
3) Scars

A. Surgical scar

- Name of scar (see the diagram)
- Healing either by Primary or secondary intention.
- Complication : Sinus formation, keloid, pigmentation, delayed healing, bleeding, infection, incisional hernia (impulse on cough)

B. Caustic Scar

- May localize the site of the pain
- It minimizes pain (counter irritation)
- Disadvantages:
 - Bad cosmetic appearance
 - Excessive adhesions (surgery will be difficult if needed)



4) Scratch marks in obstructive jaundice



5) Striae



S. Rubra



S. Alba



S. Nigra

⊗ Etiology : due to rapid stretching of abdominal wall with rupture of elastic fibers :

⊗ Types:

- Striae rubra in cushing syndrome & steroid therapy
- Striae alba in obesity , pregnancy (striae gravidarum), tense ascites, severe dieting & healed stria rubra
- Striae nigra in pregnancy

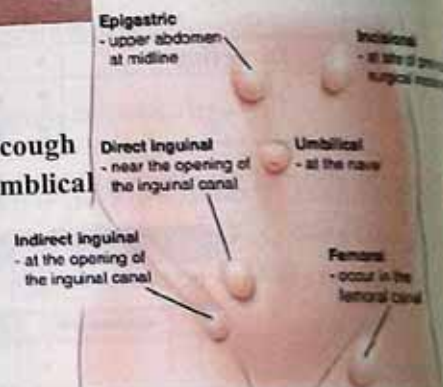
6) Subcutaneous hemorrhage: Petichae , purpura in hypersplensim & ecchymosis in LCF

7) Edema of abdominal wall : peau d'orange



8) Hernia orifices العيان واقف

- Hernia indicates: ↑ intraabdominal pressure + weak abdominal wall
- Elicited by coughing while patient is standing → expansile impulse on cough
- Types: Inguinal , Femoral , Linea ulba, Epigastric, Umbilical & paraumbilical , Incisional (old scars) hernia.



❖ Posterior Aspect = BACK العيان قاعد

- Back of the patient for swelling, scars, hair tuft in spina bifida & deformities e.g. kyphosis or scoliosis
- Back of the breast for monilial infection or masses
- Back of the Knee for Baker cyst
- Back of scrotum for TB sinus



II. Palpation



❖ شروط الفحص :

- المريض :
 - يثنى رجليه نص ثانية ← To relax superficial abdominal fascia & muscles
 - يياخذ نفس عميق من بقاء طول ما أنت بتحص
- الدكتور :
 - دفي ايديك حتى لو دافيه ولازم الدكتور يشوفك وأنت بتعملها لأن لو ايديك باردة هتعمل ← Guarding
 - طول ما ايديك على بطنه عينك على وشه

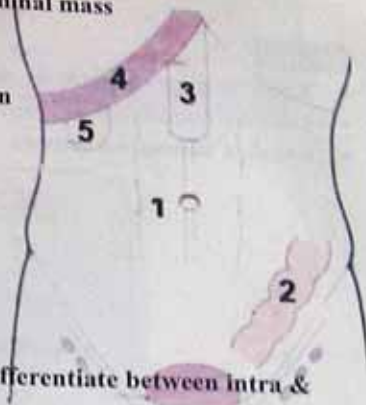
No deep fascia in abdomen

❖ Methods of palpation of abdomen:

1. Superficial (light)	2. Deep	3. Bimanual
4. Hooking	5. Dipping	6. Rolling
7. Ballotment	8. Per-rectal examination (PR)	9. Per-vaginal examination (PV)

❖ Structure normally palpable:

1. Contracted muscles of the abdominal wall may be mistaken for an intra-abdominal mass
2. Caecum and descending Colon, when it is full of gases or fluids
3. Abdominal Aorta
4. Liver edge may be felt 1-3 cm below the right costal margin on deep inspiration
5. Lower pole of right kidney especially in females with lax abdominal wall.

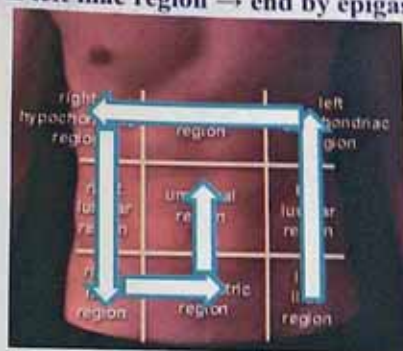
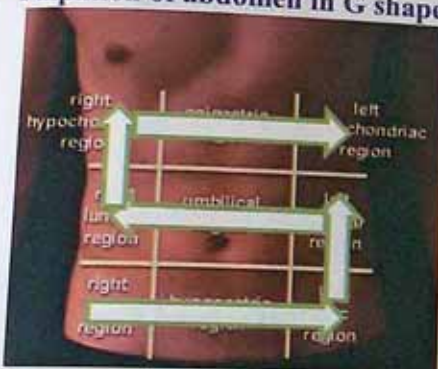


❖ Aim:

1. Gain patient's confidence
2. Detect : tenderness, hyperesthesia, temperature, guarding & rigidity
3. Detect superficial swellings e.g. lipoma , varicosities & hernia :
Ask the patient to contract the abdominal wall muscles by raising the head (differentiate between intra- & extra-abdominal swelling) and notice the swelling mobility with respiration.
4. To prepare the patient for deep palpation

❖ Method :

1. Place your extending hand flat on the patients abdomen
2. Gently press (1 cm depth) the patient's abdomen by flexion & extension of the metacarpal joints of fingers (light dipping method)
3. Slowly progress around the 9 regions either in S or G shaped palpation
 - o Start palpation away from site of complaint → end by painful region
 - o Palpation of the abdomen in S shaped manner : start in right iliac region → end by left hypochondriac region
 - o Palpation of abdomen in G shaped manner : start in left iliac region → end by epigastric region



❖ What is the difference between guarding & rigidity?

	Guarding "induced type"	Rigidity
Mechanism	Voluntary contraction of anterior abdominal wall	Involuntary contraction of anterior abdominal wall
Respiration	Disappears on inspiration	Present in inspiration & expiration
Movement with respiration	Free mobility	Limited
e.g.	During painful palpation	Peritonitis

❖ How can you examine patient with Guarding ?

- o By Nicklson's technique:

أيدي الشمال اعملها بيها Fist وأدوس على ال sternum وافحص بإيدي اليمين ,
فأنت كده خليت الحركة بتاعت ال chest تكون limited
وهيتنفس من بطنه فاجبرته انه يحرك بطنه فال guarding هيفك

❖ How to detect hyperesthesia?

- o Pinching test

o أقرص العيان قرصة خفيفة ← تلاقيه ينط من الألم

❖ How to differentiate between lipoma, hernia & varicose vein on abdomen ?

Lipoma	Hernia	Varicosities
▪ No expansile impulse on cough ▪ Slippery edge	▪ Expansile impulse on cough ▪ Reducible	▪ Expansile impulse on cough ▪ Thrill

❖ Deep palpation:

❖ Aim:

- Confirm data obtained by superficial palpation
- Localize abdominal organs (organomegaly)
- Localize abdominal masses

❖ قواعد خاصة لفحص ال Liver & spleen بالإضافة للشروط السابقة :

- استخدم ال radial side of index أو tips of fingers
- ايدك تبقى flat مع ال abdomen
- حس ال organ في النقرة ما بين ال expiration وال inspiration
- دوس بايدك لما تيجي بطنه تنزل في ال inspiration ولما يخلص اطلع بايدك بهدوء إياك والعجن!

❖ For any abdominal swelling comment on :

• Intra or extra-abdominal	• Movement with respiration	• Site	• Size
• Shape	• Skin over	• Edge	• Consistency
• Tenderness	• Pulsations	• Regional LN	

❖ Organ Palpation :

○ Liver	○ Colon	○ Para-aortic LN
○ Spleen	○ Peri-colic	○ Mesenteric LN
○ Kidney	○ Urinary bladder	
○ Gall bladder	○ Abdominal aorta	

Palpation of the Liver

❖ Never palpate liver unless percussion of upper border.

❖ Techniques :

1. Single handed method = Ordinary method: by right hand only



❖ لا تتمى تطبيق قواعد الفحص

- A. Lower border of Right lobe from Right iliac fossa in MCL upwards, lateral to rectus sheath.
 - Place your right hand flat on the right iliac fossa in mid clavicular line & feel the liver edge during the peak of inspiration (or ask the patient to take deep breath in) in one of the following position :
 - **Radial side of index finger**: Resting transversally parallel to the right costal margin & lateral to the rectus muscle
 - **Tips of fingers (middle three fingertips)** : parallel to midline & lateral to the rectus sheath.
- B. Lower border for Left lobe : from umbilicus in midline upwards

2. Enforced method :



- In thick (fatty) or rigid muscular abdominal wall
- By tips of both hands **OVER** each other (left over right)

4. Bimanual method:



- **Examination of liver**: as ordinary method but with support by left hand in lumbar region just below the right costal margin with pushing forward making liver edge more prominent.

3. Hutchinsion method = two handed method :



- In thick (fatty) or rigid muscular abdominal wall
- By tips of both hands **BESIDE** each other

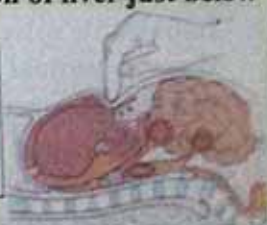
5. Hooking method:



- If liver is *not* felt by previous methods e.g. *shrunk liver*
- ❖ The examiner stands near the head of the patient
- ❖ Place both hands, side by side under the right costal margin
- ❖ Fingers of both hands are flexed as a hook
- ❖ Ask the patient to take a deep breath & feel the liver edge.

6. Dipping method in *tense ascites* with flexion at the metacarpo-phalangeal joint at the region of liver just below the right costal margin.

- إعمل خبطة واحدة (أو خبطتين) من ال MCP Joints وخلي إيدك دايسة بعد الخبطة ما تشلهاش
- لو حاجة خبطت في إيدك ← Palpable
- عيب هذه الطريقة : ما تطلعش Data



Palpation of the Spleen

❖ Techniques :

1. Single handed method = Ordinary method: by right hand only



❖ لا تنسى تطبيق قواعد الفحص

- Place your right hand flat on the right iliac fossa and direct your hand towards the left costal margin & feel the edge of enlarged spleen during the peak of inspiration (or ask the patient to take deep breath in) in one of the following position :
- **Radial side of index finger:** parallel to the left costal margin
- **Tips of fingers (middle three fingertips) :** perpendicular to the left costal margin

2. Enforced method :



- In thick (fatty) or rigid muscular abdominal wall
- By tips of both hands **OVER** each other (left over right)

3. Hutchinsion method = two handed method :



- In thick (fatty) or rigid muscular abdominal wall
- By tips of both hands **BESIDE** each other

4. Bimanual method:



- Left hand supporting lower left rib cage & press forward.
- And continue examination as single handed method by middle three fingertips from Right iliac fossa to left costal margin in the following position:
- ❖ **In supine position** (with flexed knees & hips)
- ❖ **IF NOT FELT** : do bimanual technique In **Right lateral position** (with flexed knees & hips)
- ❖ **IF NOT FELT** : impalpable spleen (don't not say: not enlarged)

❖ N.B.:

- **Palpable spleen** means, spleen enlarged 3 times or more than normal size.
- **Impalpable spleen** means, spleen enlarged less than 3 times its normal size → Further evaluation needed to detect splenomegaly.

5. Hooking method: الدكتور هيقيف على شمال العيان



- For Impalpable spleen.
- ❖ The examiner stands near the head of the patient on left side
- ❖ Place both hands, side by side under the left costal margin
- ❖ Fingers of both hands are flexed as a hook
- ❖ Ask the patient to take a deep breath & feel the spleen.

6. Dipping method in *tense ascites* with flexion at the metacarpo-phalangeal joint at the region of spleen just below the left costal margin.

- Technique : as in liver

❖ **Comment on liver:**

1. Site: in right hypochondrium
 2. Size: normally not felt below costal margin
 3. Enlarged: in patient finger-breadths (or cm) in midline for left lobe & below costal margin in MCL for right lobe
 4. Shrunk: liver cirrhosis & Bilharzial fibrosis
 5. Border = Edge: normally felt as resistance
 6. Sharp: liver cirrhosis & Bilharzial fibrosis
 7. Rounded: congenital, inflammatory, infiltration
 8. Surface: normally smooth حركة اليدك على سطحه في دائره واسعة وحس السطح
 9. Smooth: congestion, inflammation, infiltration, cirrhosis
 10. Irregular: liver cirrhosis if macro-nodular &
 11. Nodular: malignancy
 12. Consistency: normally soft
 13. Soft & enlarged: congenital, inflammation & infiltration
 14. Firm: Bilharziasis
 15. Hard: malignancy
 16. Cystic: amebic abscess & hydatid cyst
 17. Tenderness: Test to exam tenderness: fist test in non-palpable liver
- إيدك الشمال مفرودة على سطح ال liver
 اخبط بقبضتك اليمين عليها خبطة خفيفة وبص على وش العين وفارن مع الناحية المقابلة
- Congestion, inflammation & malignancy
 - 7. Pulsation: TR, TS, hemangioma
 - **Examination for pulsation by bimanual examination:** put on hand on the liver anteriorly and the other hand at the back, ask the patient to hold his breath and feel for pulsation.

❖ **Characters of liver:**

- Intra-abdominal organ
- Right hypochondrium
- Moves up & down with respiration
- Hand can't reach the upper border
- Dull on percussion & it's dullness is continues with liver dullness

❖ **Example of a FULL COMMENT on the liver:**

- Intra-abdominal swelling moves up & down with respiration
- Right Lobe: In right hypochondrium, Felt ... cm (or finger breadth) below right costal margin in right mid clavicular line
- Left Lobe: may extend to epigastrium Felt ... cm (or finger breadth) below xiphisternum in the midline
- Not warm, not tender, not pulsatile
- Firm in consistency, sharp border with smooth surface
- Hand can't reach the upper border,
- Dull on percussion & its dullness is continues with liver dullness

❖ **Comment on Spleen:**

- 1) Site: In left hypochondrium
 - 2) Size: Not felt below costal margin: normally or impalpable spleen
 - 3) If palpated: enlarged at least 3 times
 - 4) If enlarged & doesn't cross the midline (or umbilicus): patient's finger breadth
 - 5) If crossing midline → huge spleen
 - 3) Border = edge: Bilharziasis has a rounded anterior edge with one or multiple notch
 - 4) Surface: smooth in Bilharziasis
 - 5) Consistency: Soft: in endocarditis, Malaria & septicemia Firm in Bilharziasis
 - 6) Tenderness: In inflammation: typhoid & Brucellosis, TB (miliary), SBE, infarction
 - 7) Pulsation: splenic arterio-venous fistula
 - 8) Pitting sign = Mofti's sign: in chronic myeloid leukemia
 - 9) Notch in the antro-medial border
- نوس بآل thumb على ال Spleen وخليك دابس شويه ثم امش بايدك على سطح ال spleen لو لقيت حفرة يبقى positive
- Embryologically, the spleen is formed by fusion of splenules. The notch is formed at the site of their fusion

❖ **Characters of spleen**

- Intra-abdominal organ
- Left hypochondrium oblong in shape
- Moves up & down with respiration
- Hand can't reach the upper pole as you can't insinuate fingers between costal margin & the organ
- Dull on percussion & it's dullness is continues with Traub's area dullness "no band of resonance"
- Palpable notch in the antro-medial border (pathognomonic)
- Does not fill and can't be pushed in renal angle
- No ballottement "anterior ballottement only"

❖ **Example of a FULL COMMENT on the spleen:**

- Intra-abdominal swelling moves up & down with respiration, oblong in shape in left hypochondrium extending to:
- Felt ... cm (or finger breadth) below left costal margin in left mid clavicular line or
- Huge i.e. crossing midline
- Not warm, not tender, not pulsatile
- Firm in consistency, sharp anterior border with smooth surface
- Hand can't reach the upper pole as you can't insinuate fingers between costal margin & the organ
- Dull on percussion & it's dullness is continues with Traub's area dullness "no band of resonance"
- Palpable notch in the antro-medial border
- Does not fill and can't be pushed in renal angle
- No ballottement "anterior ballottement only"

❖ What are the causes of ... ?

		Splenomegaly	Hepatosplenomegaly	Hepatomegaly
Infections	Bacterial	- Paratyphoid - Typhus - T.B. - Anthrax	- Typhoid - Brucellosis - Syphilis - abscess	- Leptospirosis - Pyogenic cholangitis - Pyelophlebitis - Portal pyemia
	Parasitic	Chronic malaria	- Bilharziasis - Hydatid cyst - Kala azar	- Amebic hepatitis - Toxoplasmosis
	Viral	Psittacosis	Infectious mononucleosis	- Viral hepatitis - CMV - Herpes simplex
Tumors	Benign	Lymphangioma	Hemangioma	- Metastasis - Hepatoma - Holangioma
	Malignant	- Fibrosarcoma - Waldenstrom macroglobulinemia	Malignant lymphoma	- Wilson's disease - Hemochromatosis - Fatty liver e.g. Reye's, DM - Lipid storage disease - Glycogen storage disease (Von Geirkes)
Metabolic		- Gaucher's disease - Porphyria - Rickets	Amyloidosis	Megaloblastic anemia
Blood		- Thrombocytopenia - Thalassemia major	- Chronic myeloid leukemia - Hemolytic anemias - Polycythemia rubra vera (PRV) - Myelofibrosis	
Circulatory		- Portal vein occlusion : - Tumour - Thrombophlebitis	Portal hypertension	- Chronic venous congestion - Congestive heart failure - Pericardial effusion - Constrictive pericarditis
Congenital		Cysts of spleen		- Reidel's lobe - Polycystic disease
Collagen diseases			- Felty's syndrome - Still's disease - Sarcoidosis	

• What are the causes of palpable liver without hepatomegaly ?

- Emphysema , subphrenic abscess & occasionally in normal persons

❖ What are the causes of tender splenomegaly ? TIBS

- Typhoid , typhus , TB (military)
- Infective endocarditis , Infectious mononucleosis , Infarction
- Brucellosis & Septicemia

❖ Causes of huge splenomegaly; spleen which crosses the midline :

- Portal hypertension (bilharziasis)
- Thalassemia major
- Chronic myeloid leukemia
- Chronic malaria
- Myeloproliferative disorders (Polycythaemia rubra vera & Myelofibrosis)
- Kala azar
- Gaucher's disease
- Splenic sarcoma

❖ Causes of enlarged tender liver :

1. Malignancy
2. Infection : amebic hepatitis & abscess , viral hepatitis, pyogenic abscess
3. Congestion : heart failure , constrictive pericarditis

❖ Causes of nodular liver :

- Bilharzial with coarse nodularity
- Post necrotic cirrhosis (post hepatitis)
- Malignancy
- Syphilis Gumma (heparlobatum)
- Hydatid disease

❖ What are the causes of absent splenic notch ?

- Congenital, infarction, adhesions , tumors.

❖ What are the causes of multiple splenic notch ?

- Congenital , Multiple infarction of spleen

• Why does enlarged spleen descend obliquely towards the umbilicus?

- Towards right iliac fossa : due to presence of phrenicocolic ligament & it's axis .

• When does enlarged spleen descend towards left iliac fossa?

- ❖ Absent phrenicocolic ligament : embryological
- ❖ Resection : hemicolecotomy
- ❖ Infiltrated phrenicocolic ligament : by malignancy
- ❖ Weak ligament : visceroptosis
- ❖ Huge spleen

Palpation of the Kidney

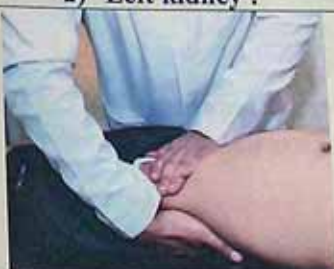
❖ Definitions :

- ❖ Renal angle : angle between last rib and lateral border of sacrospinalis muscle
- ❖ Loin : lies between last rib and iliac crest and from vertebral column to flank
- ❖ Flank : lateral aspect of the loin
- ❖ Examination of the renal angle from back while the patient is sitting:

Renal angle	Normally	Abnormally
- Inspection - Skin : Normal - Shape :concave (Empty)		- Skin : Ulcers, scars ⊗ Shape: - Fullness : swelling within renal capsule - Bulge: swelling outside renal capsule - Swelling
- Palpation - No swelling - No tenderness		⊗ Tenderness : - Thumb pressure test - Costo-vertebral percussion - Murphy's kidney punch
Percussion	Resonant	Dullness
Auscultation	Nothing	Bruit maybe heard in case of renal artery stenosis (Can be heard commonly anteriorly at transpyloric plane)

❖ Techniques:

A. Bimanual method

1) Right kidney :	2) Left kidney :
	
The Right hand is placed anteriorly in the Right lumbar region while the Left hand is placed posteriorly in the right loin	The Left hand is placed anteriorly in the left lumbar region while the Right hand is placed posteriorly in the left loin.
- بعد ما تحط ايديك , اضغط بايديك الإثنين بأقصى ما تستطيع - ثبت ايديك على كده ثم اجبر ال kidney انها تتحرك وذلك بأن تقول للعيان خذ نفس جامد وخرجه , لو كبيرة هتحتها بتتحرك طالعاه نازلة	

❖ Palpation of the left kidney on the left side:

- Move to the patient's left side.
- Place your right hand behind the patient in the left loin & your left hand anteriorly in the left lumbar region.
- The patient is asked to take deep breath while approximating both hands firmly



B. Ballotment technique (only if kidney is enlarged):

- Your hands at the same previous places, try to push the kidney with one hand & receive it with the other
- حط إيديك في الوضع السابق (من غير ما تضغط جامد)
- تخبط ال Kidney من فوق ← تنزل لتحت ← تطلع تخبط في إيديك تاني
- تخبط ال Kidney من تحت ← تطلع لفوق ← تنزل تخبط في إيديك تاني
- تسمى على اسم الأيد اللي تحس:

← اليد اللي حسنت اللي فوق Anterior Ballotment

← اليد اللي حسنت اللي تحت Posterior Ballotment

:

- ❖ Old school : name according to the receiving hand (which feel)
- Kidney : ballot anteriorly & posteriorly , Spleen : ballot anteriorly but not posteriorly
- ❖ Recent school : ballotment if you feel both anterior & posterior
- Kidney : ballotable, Spleen : non ballotable

❖ N.B.:



- ✦ Thumb pressure test: apply pressure with the thumb to Costovertebral angle
- ✦ Costovertebral percussion: using ulnar aspect of the hand strike the patient flank.
- ✦ Murphy's kidney punch :
 - While the patient is sitting, examine for tenderness in the renal angle by using the ulnar border of the right fist, percuss over the dorsum of the left hand placed on the renal angle; with comparing both sides.
 - It may allow distinction between renal tenderness or tender abdominal wall or muscles of the back.

❖ **Comment on kidney:**

- 1) Site:
 - ⊗ In lumbar region
 - 2) Size :
 - ⊗ Causes of kidney swellings : hypernephroma, polycystic kidney, perinephric abscess & hydronephrosis
 - 3) Border = edge : Rounded
 - 4) Surface : bossy , irregular in malignancy
 - 5) Consistency:
 - ⊗ Cystic in polycystic kidney
 - 6) Tenderness :
 - ⊗ Causes of tender renal swellings: Pyelonephritis , renal infarction , perinephric abscess & hydronephrosis.
- ❖ **Can you palpate normal kidney ?**
- Yes, in :

Very thin person	Lower pole only	Right kidney only
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❖ **What are the difference between spleen & kidney?**

• **ليه قولت عليها Kidney مش Spleen** أو **ليه قولت عليه Spleen مش Kidney** ؟

	Spleen	Kidney
▪ Notch	○ Yes	○ No
▪ Anterior & Posterior Ballotement	○ Non ballotable ○ Anterior only	○ Ballotable ○ (anterior & posterior)
▪ Anterior	○ It is difficult to insinuate my hand between the swelling & the costal margin & if so I can't reach the upper border	○ I can insinuate my hand between the swelling & the costal margin & ○ I can reach the upper border
▪ Posterior	○ Can't be pushed to renal angle	○ Can be pushed to renal angle
▪ Percussion	○ Dull all through	○ Band of resonance
▪ Surface	○ Smooth	○ Bossy
▪ Edge	○ Sharp	○ Rounded

Palpation of the Gall bladder

❖ **Normally GB cannot be felt, felt only if enlarged.**❖ **Examination of GB is palpated for swelling & tenderness**

1. Tenderness: Murphy's sign = acute cholecystitis :
 - Compress your right fingertips below the right costal margin at the site of the GB & ask the patient to take deep inspiration & observe his face & breathing :
 - Continue breathing → normal
 - Pain & can't continue inspiration → cholecystitis



2. Swelling :
 Site : right hypochondrium
 Technique : single handed method by deep palpation as liver
 Causes of GB swelling :

Painless :	Painful:
○ Tumour of GB ○ Cancer head of pancreas : → Courvoisier's sign: ▪ Palpable distended gall bladder in pancreatic cancer	○ Cystic duct stone : mucocele , pyocele ○ Torsion ○ Pericholecystic abscess

❖ Characters of Gall Bladder swelling:

○ Intra-abdominal organ in Right hypochondrium ○ Pyriform or rounded in shape ○ Inseparable from liver	○ Moves with respiration (up & down) ○ Cystic in consistency, hard in malignancy ○ Dull on percussion & continuous with liver dullness
--	--

❖ Differential Diagnosis : accessory hepatic lobe (Riedel's lobe)

Palpation of the Urinary bladder

❖ Normally UB is not palpable, felt only in retention

❖ Site: middle line, never cross umbilicus

❖ Technique :

- Deep palpation from the umbilicus downwards by tips of fingers or by radial aspect of index finger of left hand

❖ Characters of distended UB "retention of urine":

• Pelvi-abdominal swelling i.e. Has no lower border • Oval in shape, smooth, firm, regular • Direct pressure on it produces a desire to micturate	• Immobile, in suprapubic region • Positive fluid thrill • Dull on percussion
---	---

❖ Differential Diagnosis:

○ Gravid uterus	○ Ovarian cyst	○ Any uterine mass e.g. mesenteric cyst
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Palpation of the Colon

❖ Technique: rolling technique in 2 perpendicular directions (horizontal then vertical) :

- حط إيدك الإثنين فوق بعض (الإيد الشمال اللي فوق تدوس والإيد اليمين اللي تحت تحس)
 ○ اعمل rolling بال tips of fingers

A. Coecum :



- ❖ Site : rolling technique in right iliac fossa
- ❖ Normally coecum may be felt
- ❖ Characters of coecum :
 - Soft, rounded swelling with indistinct borders
- ❖ Differential diagnosis :
 - Tumors of coecum

B. Pelvic colon "sigmoid colon"



- Site: rolling technique in left iliac fossa
- Normally the pelvic colon may be palpable in thin patient or irritable bowel syndrome (spastic colon)
- ❖ Characters of pelvic colon:
 - Oblong sausage-shaped (about 12 cm length)
 - Does not move with respiration, parallel to the inguinal ligament
 - Movable from side to side (but not vertically)
 - Gurgling sensation on pressure
- Differential Diagnosis; mass in left iliac fossa:
 - Commonest in children : iliac adenitis
 - Commonest in middle age : Bilharzial pericolic mass
 - Commonest in old age : cancer colon

Palpation of the Bilharzial pericolic mass

❖ **Site** : left iliac fossa, **Technique** : rolling technique

❖ **Characters** :

- Moves from side to side but not up down
- Oval in shape
- Firm, ± tender

❖ **Pathogenesis** :

- Heavy deposition of ova in the subserous area → Bilharzial reaction → large subserous mass (pericolic mass)
- If deposition of ova submucous → Bilharzial reaction → intestinal polypi

❖ **Clinically** the mass can't be felt by per-rectal examination

❖ **No need for excision of the mass because:**

- It is not precancerous
- Does not cause obstruction

❖ **Investigated** mainly by endoscopy

❖ **Significance** : Associated with bilharzial polyposis:

- Ask the patient for bleeding per rectum
- Examine the patient for : toxic clubbing
- If so, treated by polypectomy

❖ **Differential Diagnosis**: mass in left iliac fossa.

Palpation of Para aortic Lymph node

❖ **Significance** : if felt in malignant cases, signifies preoperative inoperability

❖ **Technique**: **Rolling technique**: see chapter lymphadenopathy

خط إيدك الإثنين فوق بعض (الإيد الشمال اللي فوق تدوس والإيد اليمين اللي تحت تحس)
عمل rolling بال tips of fingers

Para-aortic LN

- **Site** : : umbilical & epigastric regions along lateral border of aorta i.e. on the left of a line extending from xiphisternum to umbilicus
- **Characters** : site, size, shape,
- **Significance** : if felt in malignant cases, signifies preoperative inoperability

Palpation of the abdominal aorta

❖ **Technique**: by deep palpation using two handed method to the midline above & lateral to umbilicus.

Palpation of the ascites: Transmitted thrill (fluid thrill) in tense ascites; see later

Per-rectal (PR) examination

1. **Patient position** : left lateral position (best), Knee-elbow position (maybe)

2. **Inspection** : separate buttocks carefully inspect perianal area and anus for :

- | | | |
|--|--|---|
| ▪ Pruritis ani (signs of inflammation) | ▪ External piles or fistula or pilonidal sinus | ▪ Anal warts, fissure,
▪ Ask patient to bear down for prolapse |
|--|--|---|

3. **Palpation** :

☞ Put a lubricant on gloved index finger of Right hand

☞ Ask patient to relax & introduce your finger gently & Rotate finger 360° in anal canal for :

- Thickening or irregularity in wall
- Tone of anal musculature & sphincters
- Then pass finger into rectum with left hand placed on patient Right hip & latter in suprapubic position :

☞ On withdrawing finger look at it for blood, mucous, pus

4. **Value of PR** : to help in diagnosis of the following conditions :

- | | |
|--|--|
| 1. Alimentary problems e.g. | 2. Genito - urinary problems e.g. |
| • Anal irritation or pain, tenesmus or rectal pain | • Hematospermia & epididymo-orchitis |
| | • Gynecological examination in virgins |

3. **Miscellaneous problems** :

- In the search for a cause of backache, root pains in legs or diffuse bone pains



III. Percussion

- ❖ Type : percussion of abdomen by light percussion except upper border of liver by heavy percussion
- ❖ Values :
 - Defining boundaries of abdominal organs & masses
 - Detection of ascites

Percussion of the liver

Percussion of the liver		
❖ Border	A. Upper border	B. Lower border
❖ Aim = Value = importance of the border:	○ Liver span: see below ○ To differentiate ptosed liver (emphysema) from enlarged liver	○ Shrunk liver ○ Soft liver ○ Tenderness ○ Rigid abdomen ○ Light percussion
❖ Type of percussion	○ Heavy percussion confirmed by tidal percussion	
❖ line	○ Right MCL	○ Right lobe : in the RT MCL stating from right iliac fossa upwards ○ Left lobe: in the midline, starting from just above the umbilicus upwards
❖ In percussion of the lower border percuss upwards till reaching dullness of the liver, When you reach it, continue percussion , till you reach the upper border, to prove that is dull on percussion and it's dullness continuous with normal hepatic dullness		

By heavy percussion:

Upper border



By light percussion: lower border of the :

Right lobe



Left lobe



❖ To determine the liver span:

→ Definition : the vertical distance in cm between the dullness of the upper & lower border of liver in MCL:

- Determine the upper border of liver dullness by heavy percussion confirmed by tidal percussion
- Determine the lower border of liver dullness by light percussion or by palpation.

→ Results :

- Normal span: 8-16 cm in the right MCL.

❖ Normal liver span in midline : 4-8 cm, but can't be detected clinically

▪ Abnormalities:

- The span is increased in case of hepatomegaly.
- The span is decreased in case of shrunken liver (liver cirrhosis).
- The upper border of liver dullness is displaced downwards in case of low diaphragm due to COPD



II. Impalpable splenomegaly

- A. Percussion of Traube's area:



- From the center of Traube's area to left mid axillary line at the level of 9th intercostal space:
 - If you found no dullness till MAL → no splenomegaly
 - if you found dullness before MAL → splenomegaly
- B. Castell's technique
- C. Nixon's technique

A. Castell's technique = splenic percussion sign: للإمتيااااا

- Technique:
 ➤ Percuss left 8th, 9th or 10th intercostal spaces along left AAL twice :

- Once in expiration
- Once in deep inspiration

- ☞ Normally : resonant

- ☞ Abnormally : splenomegaly

- In expiration : resonant

- In deep inspiration : dull =(lower pole of the spleen is displaced inferiorly & medially)

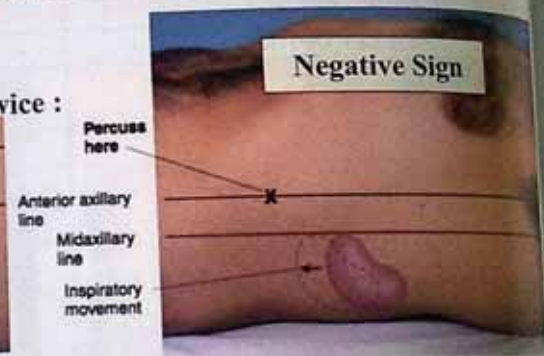
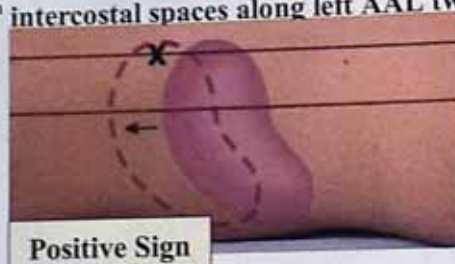
B. Nixon's technique: لأوائل الدفعه

- ☛ The patient lies in right lateral decubitus

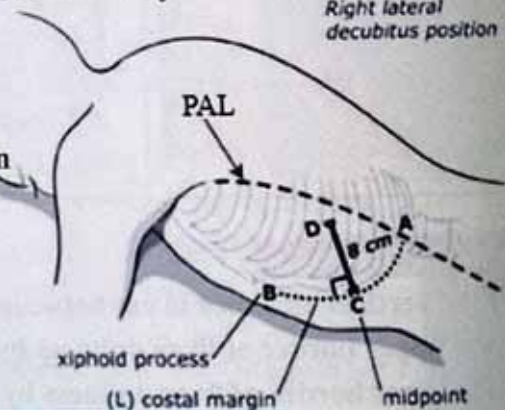
- Percuss with your finger parallel to the left costal margin from the PAL to the midpoint of the left costal margin crossing the splenic dullness till it disappears

- ☞ Normally : 6-8 cm

- ☞ Abnormally: splenomegaly : > 8 cm



Right lateral
decubitus position



Percussion of Urinary bladder

- **Technique :** percuss in 3 parallel lines beginning from the umbilicus:

- ☞ **Midline** : horizontal finger , percussing from just below the umbilicus downwards

- ☞ **Right flank :** horizontal finger , percussing from right costal margin downwards

- ☛ **Left flank** : horizontal finger , percussing from left costal margin downwards

-

UB characterized by :	Peripheral resonance	Central dullness	Inverted U shaped dullness
Ascites characterized by :	Dull flanks	Central resonance	U shaped dullness

Percussion of the Kidney

Back : renal angle is resonant

- Front: percuss from the corresponding iliac fossa, while your finger is horizontal over the abdomen, percuss upwards, when you reach the dullness of the kidney swelling, continue percussion upwards till the upper border, band of resonance of the colon will be detected

Percussion of Ascites

❖ Percussion of ascites		
Mild 0.5 – 1 litre	Moderate 1.5 – 3 litre	Massive > 3 litre
❖ N.B.: Minimal ascites less than 500 cc is detected by ultrasonography Tense ascites depends on the amount & rate of accumulation (> 3000 cc)		
❖ Type		
Mild ascites	Moderate ascites	Tense ascites
❖ Idea:		
The abdomen becomes horizontal, the umbilicus is the most dependent part & the fluid collects around it	Free fluid tends to gravitate, while loops of intestine float upon	Amount makes the abdomen under tension
❖ Technique:		
Percussion in Knee elbow position	Shifting dullness	Transmitted thrill (fluid thrill)
		
1. خبط على الـ umbilicus والعين نايم resonant هتلاقىها 2. قول للعين اسجد ثم هم على كوعك (knee-elbow) وخبط على الـ umbilicus مرة ثانية هتلاقىها بقت dull	ابدأ من الـ umbilicus نازلاً لأسفل حتى تصل للـ dullness علشان نجيب الـ upper border اطلع فوق سنة واقرب ايدك خليها عمودية واعمل percussion للناحية اليمين او الشمال ثم خبط حتى تصل للـ dullness ثبت ايدك في هذا المكان وخلي العين ينام على جنبه الثاني وانتظر دقيقة ثم خبط في نفس المكان مرة أخرى لو لقيتها اصبحت resonant يبقى في ascites ثم كرر نفس الشيء على الناحية الأخرى علشان تتأكد ان ده free fluid مش encysted	1. حط ايد العين بسيفها على منتصف البطن وقول له دوس جامد وفاندتها انك: To damp an impulse which may be transmitted through fat of abdominal wall Make the abdomen more tense 2. ثم اعمل flickering بايد وحس بال palm بتاع بالايه الثانية
❖ N.B.:		
• How to increase the accuracy of detection of minimal ascites using stethoscope? • by puddle sign	❖ Percussion of upper border of ascites from umbilicus or below the level of dullness of organomegaly ❖ Variations of technique : • Percuss the upper border in 3 parallel → U shaped • Some doctors do not perform the 1 st step (upper border) • Shifting dullness or shifting resonance ❖ Causes of unilateral shifting dullness: • Ovarian cyst, intestinal obstruction & encysted ascites	• Not specific for ascites may be found in ovarian cyst & huge hydatid cyst

IV. Auscultation

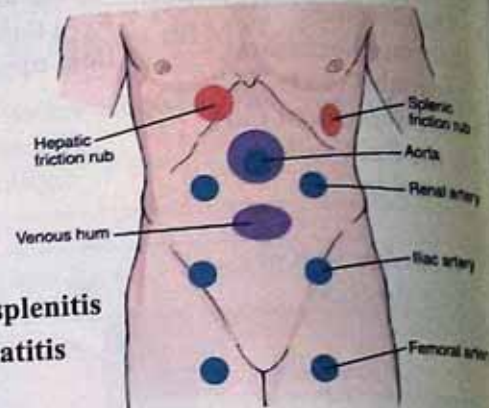
- Pulmonary area** : for pulmonary hypertension (Billharzial cor-pulmonale)
- Succussion splash**: to diagnose gastric stasis as in pyloric obstruction
- Value of Succussion splash** : Hydropneumothorax & Gastric stasis as in pyloric obstruction

3. Vascular sounds:

	Arterial bruit	Venous hum
Timing	Only during systole	Continuous "systolic & diastolic"
Character	High pitched	Low pitched
Heard By	Diaphragm	Cone
Heard at	- Renal angle - Trans-pyloric line	Epigastric area; between xiphisternum & umbilicus
Variation with inspiration	No change	Increased with inspiration
Cause	Renal artery stenosis	Portal hypertension "Kenawy sign"

4. Intestinal sounds (borborygmi):

- Start abdominal examination by auscultation of intestinal sounds before palpation, because excessive maneuver in abdomen leads to irritation of intestine & increases rate of sounds
- Character: low pitched gargles of variable frequency (5-30 /min)
- Heard by cone, at : right & lower to the umbilicus
- Increased in : diarrhea & early intestinal obstruction
- Absent in : paralytic ileus & peritonitis
- Auscultate for peristalsis bowel sounds for at least 3 minutes before deciding that they are absent



5. Friction rubs :

- Splenic rub : in Left hypochondrium, due to splenic infarction & perisplenitis
- Hepatic rub: in Right hypochondrium, due to hepatic tumor & perihepatitis

6. Pregnancy:

- Fetal heart sounds
- Uterine soufflé: flow of blood in uterine artery.

7. Scratch test : auscultatory method = Macleod method: for soft hepatomegaly or splenomegaly or urinary bladder

- Idea : sound transmitted better in solid media
- Technique for liver as an example:



❖ How to detect soft hepatomegaly ?

- Percussion of lower border
- Scratch method

خط السماعه على ال right costal margin
خريش بايديك أو بقلم في خطوط متوازية صاعداً لأعلى
لما تلاقى الصوت اللي انتة سامعه بالسماعه على فجأة , خط نقطة ثم وصل هذه النقطة هو ده ال lower border بتاع ال liver

	Position of stethoscope	Scratching movement	Increased intensity of sound detects
Liver	Xiphisternum or above right costal margin	From right lower quadrant towards the right costal margin along MCL	Lower border of liver
Spleen	Above left costal margin	From left lower quadrant towards the left costal margin along MCL	Lower border of spleen
Urinary bladder	Supra-pubic area	Along the vertical midline from the umbilicus downward	Upper edge of the urinary bladder

8. Puddle sign: for detection of minimal ascites :

- The patient lies prone for 5 minutes
- The patient lies in the knee-elbow position
- Place the stethoscope on the most dependent part of the abdomen (Place stethoscope at umbilicus)
- The nearby flank is percussed repeatedly by light flicking of constant intensity
- Gradually move the stethoscope towards the flank opposite the percussion
- An increase in the intensity of the sound hear by the stethoscope indicated that there is a fluid level

❖ Revision : Signs of Liver cell failure

Cellular decompensating

❖ Vital signs

Low grade fever



Tachycardia



Decrease diastolic pressure



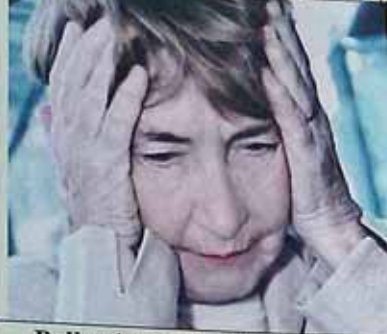
Wasting of muscles

Temporalis Muscles



❖ Head & neck

Hepatic encephalopathy (SAD)



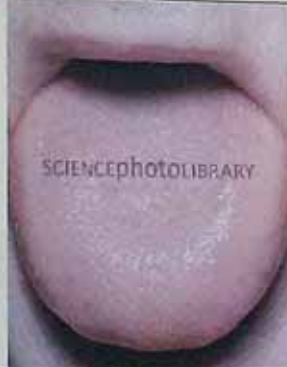
Fine silky hair اذعو له بالرحمة لوسمحت



Hepatocellular Jaundice



Pallor in conjunctiva, lips, tongue



Central cyanosis in tongue



Foetor hepaticus "rotten apple"



Spider naevi



Xanthelasma



Endemic parotitis + Paper money skin



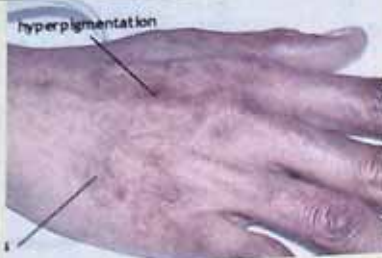
Congested neck veins



Spider man appearance



❖ Upper & Lower limbs

❖ Upper & Lower limbs		Palmar or tendon xanthomata
Asterixis	Palmar erythema	
		
Pallor	Pigmentation	Paper money skin
		
Capillary pulsation & Water hammer pulse		Cyanosis & Cyanotic clubbing
		
Changes in Nail :		
Koilonychia	Leukonychia	Muehrcke's nails
		
Dupuytren Contracture	Bilateral pitting lower limb edema	Spider naevi
		
Scratch Marks	Subcutaneous hemorrhage	
		

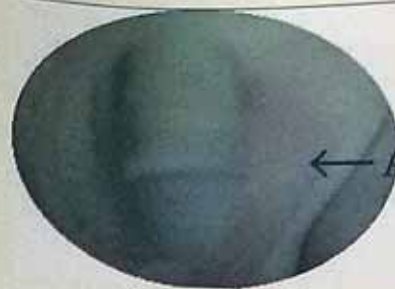
Ascites



❖ Local examination
Feminine distribution of
supra-pubic hair



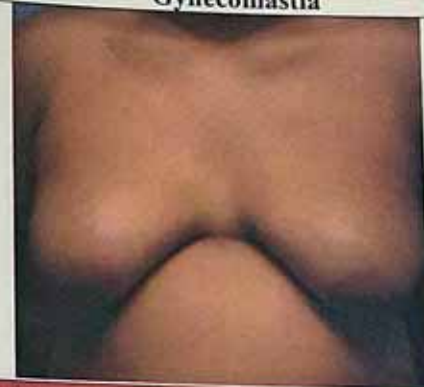
Testicular atrophy



Atrophy of female breast



Gynecomastia



Vascular decompensating : portal hypertension

Splenomegaly is the surest sign of
portal hypertension = congestive
splenomegaly



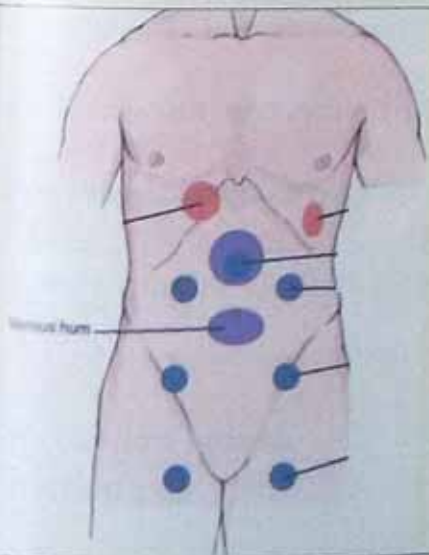
Bleeding varices :
Oesophageal varices
(history of
hematemesis)



Porto-systemic anastomosis
"caput medusa "



Venous hum



Ascites



Diagnosis Of Abdominal Case

❖ Etiological & pathological diagnosis = SCHEME of the abdomen

Case	1) Malignancy	2) Hepatitis "post hepatic cirrhosis"	3) Bilharziasis "pure B fibrosis"	4) Mixed "mixed B & hepatitis cirrhosis"
History	- Constitutional symptoms : - Marked - Short duration - Most of them present.	- History of blood transfusion - Fever - Jaundice - Dark urine & pale stool - Admission to fever hospital	- B dysentery - Investigation for B "stool analysis" - Received treatment - Anti B drugs - Contact with channel water	Hepatitis & Bilharziasis
General examination	Cachexia = emaciation + pallor	Signs of LCF	No LCF	Signs of LCF
Local examination	- Data of malignancy : - Hard consistency - Irregular surface - Rounded edge - tender	- Data of cirrhosis : - Firm in consistency - Smooth or Nodular surface - Sharp edge - Shrunk liver	- Data of fibrosis : - Firm in consistency - Smooth surface - Sharp edge - Shrunk liver	Data of cirrhosis.
Investigations	- Tumor marker : ↑ alpha fetoprotein - Biopsy	Hepatitis markers	Stool analysis : viable ova "hatching test"	Hepatitis markers & Stool analysis

❖ Anatomical diagnosis by clinical examination & investigation (the best is Ultra-sound):

- Splenomegaly
- Hepatosplenomegaly
- Hepatomegaly

❖ Functional diagnosis by clinical examination & investigation:

A. Compensation :

- Compensated
- Decompensated :
 - Cellular decompensation: see before
 - Vascular decompensation: see before

B. Complications & how to suspect clinically :

- Hepatic encephalopathy (SAD):**
 - Sleep disturbances (somnolence & inverted sleep rhythm),
 - Speech disturbances (slurred speech or monotonous speech)
 - Apathy, Asterixis
 - Disturbed personality & behavior
 - DCL with disorientation for time, place & person.
- Hypersplenism:** anemia, infection & bleeding tendency
- Peritonitis "spontaneous bacterial peritonitis"** : fever, abdominal pain & tenderness, refractory ascites
- Malignancy:** see before.

E.g.

- This is a case of bilharzial periportal fibrosis combined with post-hepatic cirrhosis with shrunken liver, enlarged spleen & moderate ascites. The condition is decompensated both cellular & vascular & not complicated
- A case of hepatosplenomegaly, mostly post hepatitis liver cirrhosis with portal hypertension, no manifestation of liver cell failure
- A case of splenomegaly for differential diagnosis mostly due to liver cirrhosis with shrunken liver with portal hypertension & manifestation of liver cell failure

Summary Of Abdominal Examination

• Vital signs	• General overview	• Systemic overview	• Other systems except that of local exam
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I. General examination:

• Local examination : Abdominal

II. Inspection of the Abdomen = Anterior + Back

Anterior aspect:

2 General :

1. Contour of the abdomen
2. Movement with respiration

❖ 7 in Midline

1. Sub costal angle
2. Divarication of recti
3. Umbilicus (5 S + 2 D)
4. Epigastric pulsation
5. Visible peristalsis
6. Supra-pubic hair
7. External genitalia

❖ 8 on Sides:

4 :

1. Breast
2. Dilated vein
3. Edema of abdominal wall
4. Hernia

4 :

5. Scars
6. Scratch marks
7. Striae
8. Subcutaneous hemorrhage

☞ Posterior aspect : back of the patient, breast, knee & scrotum

III. Palpation:

A. Superficial palpation : tenderness , guarding , ... etc.

B. Deep palpation :

1. Liver for:	2. Spleen	3. Kidney
▪ Lower border of right in MCL		
▪ Lower border of left lobe in ML		
4. Gall bladder	5. Urinary bladder	6. Colon
7. Para-aortic LN & mesenteric LN	8. Abdominal aorta	9. Ascites (transmitted thrill)

10. Per-rectal examination (PR)

IV. Percussion:

1. Liver :	2. Spleen	Urinary bladder
▪ Upper border in MCL		
▪ Lower border for right lobe in MCL		
▪ Lower border for left lobe in ML		
3. Kidney	4. Ascites: Knee – elbow, shifting dullness	

V. Auscultation

1. Succussion splash	2. Vascular sounds	3. Intestinal sounds
4. Friction rub	5. Scratch test for soft organomegaly	6. Puddle sign for minimal ascites

INVESTIGATIONS OF ABDOMINAL CASE

I. Non-Imaging:

1) Liver function tests

A. Liver enzymes :

❖ Transaminases :

- Serum glutamic Pyruvate transaminase (SGPT) = ALanin transaminase (ALT), this increases in acute liver disease & sPecific for Liver
- Serum glutamic Oxaloacetate transaminase (SGOT) = asparTate transaminase (AST) this increases in chronic liver disease & nOT specific for liver, present in heart as well.

- ❖ Gama glutamly transferase (GGT) : specific for liver disease e.g. alcoholic hepatitis
- ❖ 5-nucleotidase : increases in obstructive jaundice
- ❖ Alkaline phosphatase : increases in liver & bone disease

B. Bilirubin :

- Total : Normal : 0.2 – 1 mg%, increases in all types of jaundice
- Direct: Normal : 0.2 mg%, increases mainly in obstructive jaundice
- Indirect: Normal: 0.8 mg%, increases mainly in hemolytic jaundice

C. Plasma proteins :

❖ Normal Values:

- Total protein : normal : 7 – 9 gm %
- Albumin : normal: 4-5gm%
- Globulin : normal : 2- 3 gm%
- A/G ratio : normal 2:1

❖ Abnormally:

	Acute liver disease	Chronic liver disease
e.g.	Viral hepatitis	Liver cirrhosis
Albumin	Not affected (Long life span)	↓
Globulin	↑	↑
A/G ration	Normal	Decreased or even reversed.

D. Alpha fetoprotein: increases in hepatoma

E. Hepatitis markers

F. Prothrombin :

- Time : Normal = 12-14 sec , increases in chronic liver disease
- Concentration : Normal 100% , decreases in chronic liver disease

2) Serum fluid analysis for ascites for TB, malignant peritonitis , spontaneous bacterial peritonitis

3) Stool & Urine analysis : for viable Bilharzial ova

4) CBC:

- Pancytopenia due to hypersplenism
- Hypochromic microcytic anemia due to bleeding varices

5) ECG: Bilharzial cor-pulmonal

6) EEG: encephalopathy

7) Endoscopy :

❖ Upper GIT endoscopy : to detect :

- Esophageal varices
- Peptic ulcer, fundal varcies

❖ Lower GIT endoscopy to detect :

- Piles
- Bilharzial ulcers & polypi

Lower endoscopy



Upper endoscopy



II. Imaging:

1. Ultrasonography (US) to detect :

A. If used alone :

- Ascites , amount , free or encysted
- Liver, spleen, renal, gall bladder abnormality
- Portal circulation : diameter of portal vein (normally varies from 7 → 14 mm) & portal blood flow (Doppler)

B. US guided aspiration

C. US guided biopsy

2. Contrast X-ray:

- Barium swallow : oesophageal varices → → →
- Barium meal : peptic ulcer
- Barium enema : Bilharzial polypi

3. CT & MRI abdomen to detect malignancy.

4. Catheter:

A. Portal venography :

- Before shunt operations to ensure patency
- Was invasive but now located by digital subtraction angiography (DSA)

B. Portal manometry :

- Pre-operative
- Intra-operative
- Normal portal pressure = 7-11 mmHg

5. Liver biopsy:

A. Precautions :

- Vitamin K parenteral 5 days before biopsy
- PT concentration 100%

B. Types

- Open : wedge biopsy
- Needle biopsy : US or CT guided biopsy
- Trucut (core of tissue)
- Chiba needle

C. Indications:

- Tumour (benign , malignant)
- Chronic hepatitis (active, persistent)

D. Contraindications:

- Uncooperative patient
- Bleeding disorder
- Difficulty in determining liver location, as with ascites
- Infection of skin, pleura, right lower lung or peritoneum overlying the liver
- Suspected liver abscess or vascular lesion
- Severe extra-hepatic obstruction



CLINICAL DISCUSSION

❖ What is the definition of liver cirrhosis & causes or types ?

- Definition : diffuse chronic liver disease characterized by :
 - Degeneration , fibrosis , regeneration nodules, irreversible loss of normal structure
- Causes
 - A. Alcoholic cirrhosis
 - B. Biliary cirrhosis
 - C.

- | | |
|--|---------------------|
| ○ Cryptogenic (idiopathic) cirrhosis | ○ Cardiac cirrhosis |
| ○ Chronic hepatitis : post hepatitis cirrhosis | |
| - Either post viral hepatitis (Virus B,C,D) or autoimmune (Lupoid hepatitis , primary sclerosing cholangitis) | |
- D. Drug induced : Alpha methyl dopa, Amidarone, INH & Methotrexate
 - E. Miscellaneous :
 - Metabolic causes
 - Non-alcoholic steatohepatitis

❖ What is the definition of hypersplenism & the causes ?

- Definition : exaggeration of normal splenic function (↑ phagocytic activity) leading to destruction of one or more of the blood cells :
 - Monocytopenia, or bicytopenia or pancytopenia
- Causes :
 - Secondary : splenomegaly
 - Idiopathic : rare

❖ What are the causes of ascites & treatment ?

❖ Causes :

I. Transudative ascites :

- A. Organ failure : liver cell failure & right side heart failure
- B. Portal hypertension: **causes of portal hypertension :**
 - Supra-hepatic causes : right ventricular failure, pericardial effusion, high IVC obstruction , Budd Chiari syndrome
 - Intra-hepatic causes : liver cirrhosis , Veno-occlusive Disease, Biliary periportal fibrosis
 - Infra-hepatic causes : portal vein obstruction e.g. thrombosis or malignancy
- C. Hypoalbuminemia e.g. nephrotic & nephritic syndrome & severe malnutrition
- D. Miscellaneous e.g. myxedema , Meig's syndrome

II. Exudative ascites :

- Peritoneal disease e.g. Tb peritonitis & spontaneous bacterial peritonitis
- Pancreatic ascites due to pancreatitis

III. Chylous ascites : due to obstruction or rupture of thoracic duct e.g. Tumor or trauma

IV. Hemorrhagic ascites due to hemorrhagic blood disease

❖ Treatment :

○ Bed rest to unload liver	○ Diet : increase protein and decrease salt	○ Salt free albumin & diuretics
○ Lee ven shunt: peritoneo-venous shunt (between peritoneum & SVC)	○ Tapping	

❖ What are signs of ascites ?

1) Inspection :

- Generalized abdominal distention with fullness in flanks, umbilicus is shifted downwards & umbilicus is bulging

2) Palpation : transmitted thrill

3) Percussion : knee elbow & shifting dullness

4) Auscultation : Puddle sign

5) Ultrasonography : if clinically is not evident

6) ± fullness in Douglas pouch by P.V. examination

❖ What are the precipitating factors for hepatic encephalopathy ?

High protein diet	GIT bleeding	Constipation
Hypokalemia	Rapid tapping of ascites	Infection

❖ What is the most important treatment of hepatic encephalopathy ?

Enema	Lactulose & neomycin	Protein restriction
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FREQUENTLY ASKED QUESTIONS FOR MID & END ROUND EXAM

Test yourself!

- 1) Surface anatomy of the liver?
- 2) What are signs of ascites?
- 3) How to differentiate between lipoma, hernia & varicose vein on abdomen?
- 4) What are the causes of divaricated recti?
- 5) What are causes of bulging of umbilicus?
- 6) What makes an adult male has a feminine hair distribution (straight instead of triangular)?
- 7) What are causes of stria alba?
- 8) What are the medical causes of pruritis?
- 9) If there is a dilated vein below umbilicus, how to differentiate it's causes?
- 10) What are the causes of visible peristalsis ?
- 11) What are the aims of superficial palpation?
- 12) What are normally palpable structures in the abdomen?
- 13) What are points to comment on for palpable liver or spleen?
- 14) What are the causes of tender liver?
- 15) What are the causes of absent notch?
- 16) Causes of pulsating liver?
- 17) What is meant by huge spleen ? what are its causes?
- 18) What are causes of ascites?
- 19) What is the importance of detection of upper border of liver before palpating it's lower border?
- 20) Why spleen enlarge towards right iliac fossa?
- 21) Why we flex patient's lower limb during deep palpation of abdomen?
- 22) What are the manifestation of liver cell failure?
- 23) Causes or types of liver cirrhosis?
- 24) Causes of portal hypertension?
- 25) Treatment of ascites?
- 26) Causes of hepatomegaly?
- 27) Causes of palpable liver without hepatomegaly?
- 28) What are the causes of gynecomastia ?
- 29) Causes of parotid enlargement ?
- 30) How to decrease portal hypertension in between attacks?
- 31) All questions answered in the clinical discussion are very important.
- 32) What are the causes of generalized abdominal swelling?
- 33) How to differentiate intra-abdominal from extra-abdominal swelling
- 34) What are causes of wide subcostal angle?
- 35) What are causes of downward displacement of umbilicus?
- 36) What is caput medusa?
- 37) What are the complications of scar?
- 38) What are the causes of stria rubra?
- 39) What is the causes of visible veins on abdomen?
- 40) What are types of hernia ?
- 41) What is the importance of examination of male genitalia in abdominal case?
- 42) What is the difference between guarding & rigidity ?
- 43) What are different methods of palpation of liver and spleen?
- 44) What is the liver span?
- 45) What are the causes of multiple splenic notch?
- 46) Mofti's sign?
- 47) What is the method of renal palpation?
- 48) How can you differentiate splenomegaly from renal mass?
- 49) What is the type of percussion of abdomen?
- 50) What are the methods to detect early splenomegaly?
- 51) What are causes of vertical enlargement of spleen?
- 52) Auscultation of the abdomen?
- 53) Investigation of a liver case?
- 54) Definition of liver cirrhosis ?
- 55) Definition of hypersplenism?
- 56) Causes of splenomegaly?
- 57) Causes of tender spleen?
- 58) What is the difference between ascites & ovarian cyst?
- 59) How to diagnose gynecomastia?
- 60) Causes of pitting spleen?
- 61) Prophylaxis against portal hypertension ?

O.S.C.E QUESTIONS

- | | |
|--|---|
| • Examine spleen | • Palpate for spleen |
| • Examine liver | • Palpate liver |
| • Examine for ascites & signs of LCF in LL | • Search for ascites & signs of DM in leg |
| • Palpate the abdomen | • Palpate the abdomen for organomegaly |
| • Signs of LCF on face | • Signs of chronic liver disease |
| • Signs of LCF in hand | • Examine ascites. |

❖ **Personal history:** & There is history of contact to water canals with no history of traveling abroad.

❖ **Complaint:** Pain in left & right upper quadrants of the abdomen one week duration.

❖ **HPI:**

- The condition started 10 years ago by gradual onset and progressive course of dull aching localized pain in the left upper quadrant of abdomen, not related to position, respiration, exercise or meals. one year later, the patient developed dull aching localized pain in the right upper quadrant of abdomen, not related to position, respiration, exercise or meals. The patient sought medical advice, investigated by urine, stool analysis and abdominal ultrasonography and treated by silymarin and anti bilharzial treatment
- The patient remained symptom free till one week ago when he re-experienced dull aching localized pain in the right upper quadrants of abdomen, not related to position, respiration, exercise or meals.
- No history of abdominal swellings, abdominal distension, upper or lower GIT symptoms, hepatobiliary or urogenital symptoms, no constitutional symptoms and no L.L oedema.
- No symptoms of other system affection

❖ **Past history**

- There is past history of **Bilharziasis** Since he was 9 years old, manifested by dysentery and terminal hematuria, investigated by urine and stool analysis, treated by distocid tablets.
- No past history of blood transfusion, or past history suggesting previous attacks of acute hepatitis
- No DM no HTN.
- No past history of operations or drugs.

❖ **Family history**

- No consanguinity, No common diseases, No similar condition.

1) **General examination:**

A. **Overview**

- **Temperature** : 37
- **Blood pressure** 110/70
- **Pulse** : regular pulse, 75 /minute, average volume, no special characters, vessel wall is not felt, equal on both sides with intact peripheral pulsations, no radio-femoral delay.
- The patient is **fully conscious**, well oriented for time place and persons, average mood and memory, the patient is co-operative with average intelligence.
- the patient looks **well average. built**, No **jaundice**, **mild pallor**, **no cyanosis**.

B. **H&N**

- No parotid enlargement, no fine silky hair, no fetor hepaticus, no wasting of muscles, the skin shows no spider naevi or paper money appearance.
- Neck veins are pulsating not congested.

C. **Extremities**

- No oedema L.L.
- No clubbing, no leuconychia, no capillary pulsations and no flappy tremors.
- The skin shows no spider naevi, no palmer erythema, no paper money appearance, no S.C haemorrhage. no wasting of muscles.

2) **Local examination:**

a) **Inspection**

- The abdomen moves freely with respiration, normal abdominal contour, there is localized oval shaped swelling in the hypochondrium and left lumbar regions which decreased in size on doing rising up test, no dilated veins, regarding the skin: there is no scars, no stria no scratch marks, no sc haemorrhage, no pigmentation, no oedema in anterior abdominal wall with normal skin elasticity, no abdominal or inguinal hernias and no gynecomastia. There is wide subcostal angle confirmed by thumb test, mild epigastric pulsation, no visible peristalsis. There is mild divarication of recti. Regarding umbilicus, it is shifted downwards flat and transverse in shape no hernia no discharge. The skin shows no dilated veins no nodules no scars, pigmentation or ulcers, there is male distribution of suprapubic hair with normal genitalia, regarding back no deformity, no swelling, no scars.

b) Palpation:

❖ By superficial palpation:

- Apart from oval shaped swelling in the left hypochondrium and left lumbar regions, no other swellings, no rigidity, no tenderness, no hyperesthesia.

❖ By deep palpation:

- The liver is firm in consistency, sharp border, the right lobe is felt 3 fingers below the right costal margin in the right midclavicular line while the left lobe is felt hand breadth below xiphisternum in the midline with smooth surface. It is neither tender nor pulsating (*to be differentiated from other masses in the right hypochondrium*)
- The spleen is firm in consistency, rounded notched border, it is felt 7 fingers below the left costal margin (or it is huge in size) with smooth surface, it is not tender or pitted (*to be differentiated from other masses in the left hypochondrium and left lumbar specially left kidney*).
- Otherwise, there is **no other palpable abdominal organs.**

c) Percussion:

- Upper border of liver is in the 6th intercostals space in the right midclavicular line detected by heavy percussion with the hepatic span = 16 cm.
- There is dullness below the right costal margin and xiphisternum detected by light percussion and continuous with the hepatic dullness.
- There is dullness at the level of the umbilicus detected by light percussion and continuous with Traub's area dullness.
- No ascites detected by percussion.

d) Auscultation

- There is audible intestinal sound, no vascular sounds no hepatic or splenic rubs no succussion splash, no ascites detected by puddle's sign. the liver is detected below the right costal margin by scratch test.

? What is your diagnosis ?

- A case of hepatosplenomegaly for D.D most probably bilharzial, there is no signs of LCF and no previous attacks of bleeding with no complications *Or*
- A case of hepatosplenomegaly for D.D most probably bilharzial, there is vascular decompensation but no cellular decompensation with no complications.

? Why ?

- Hepatosplenomegaly? from local exam and sonar.
- Bilharzial? from history, urine and stool analysis and serological tests for bilharzia (rectal snip is much better).
- No cellular decompensation? from absence of symptoms and signs of LCF and LFTs.
- Vascular decompensation? from signs of portal hypertension (splenomegaly, portal venography and manometry are much better).
- Not Complicated? as there is no evidence of spontaneous bacterial peritonitis, malignancy or hypersplenism (*see diagnosis of abdominal case to know how to detect each one*).

? How would you proceed to manage this case?

A. Investigation

- Urine and stool analysis for viable bilharzial ova.
- Blood for CBC (anemia), LFT, serological tests of bilharziasis, hepatitis markers.
- ECG for bilharzial cor pulmonale.
- Barium studies for
- Ultrasonography for
- Endoscopies upper and lower for

B. Treatment

- Stop any hepatotoxic drugs
- Silymarin
- B-blockers (inderal) to decrease the portal pressure specially if there is varices detected by endoscopy (silent varices as no bleeding in the patient's story)
- Spleen can be excised if it is accused in blood cells destruction (hypersplenism)

- ❖ **Does portal hypertension of Bilharziasis cause ascites ?**
 - Portal hypertension alone rarely causes ascites; it only localizes the transudate to the peritoneal cavity, however: in hepatic schistosomiasis, ascites may develop late due to associated hypoalbuminemia.
- ❖ **What causes associated hypoalbuminemia in hepatic schistosomiasis?**
 - Decreased intake : nutritional deficiency due to poverty
 - Decreased absorption : malabsorption due to intestinal congestion
 - Decreased production : due to associated cirrhosis in mixed pathology
 - Increased loss :
 - Bleeding : bleeding esophageal varices
 - Protein losing enteropathy : colonic polyps & congestive enteropathy
- ❖ **Do you expect to find manifestations of liver cell failure in a Bilharzial liver?**
 - No, because in Bilharziasis there is only fibrosis (no degeneration, no regeneration, no loss of architecture).
 - Unless: there is associated viral hepatitis, especially HCV.
- ❖ **What are the grades of hepatic schistosomiasis (bilharziasis)?**
 - Clinical stages of hepatic schistosomiasis:
 - A. Stage I: Hepatomegaly
 - B. Stage II : Hepatosplenomegaly
 - C. Stage III : Shrunken liver & splenomegaly
 - D. Stage IV: Shrunken liver , splenomegaly & ascites
- N.B.: premature ascites means ascites occurs in stage I & II because of :
 - a) Hepatitis with bilharziasis causing liver cell failure
 - b) Bilharzial cor pulmonale
 - c) Marked malnutrition and mal-absorption caused by bilharzial dysentery
- ❖ **Can bilharziasis and hepatitis affect each other ? how ?**
 - In bilharzial patient:
 - His main problem is vascular → bleeding → not dangerous as he has good clotting factors
 - In case he got Hepatitis: Hepatitis cause LCF → destruction of clotting factor → hematemesis till
 - In hepatitis patient :
 - regeneration nodules depend on hepatic artery & portal vein for blood supply,
 - In case he got bilharziasis, hematemesis → hypovolemia & hypotension occurs → damage of regeneration nodules → LCF
- ❖ **Which type of Schistosoma is blamed to cause corpulmonale ?**
 - S. mansoni because they reach the lungs with difficulty only in the presence of portal hypertension & porto-systemic collaterals which allow the ova to bypass the liver to the lungs, however; they cause more destructive lesions than S. haematobium.
- ❖ **Why bilharzial patient more exposed to hepatitis ?**
 - a) Decreased cell mediated immunity
 - b) Blood transfusion
 - c) Injection treatment " Tartar emetic"
 - d) Virus incorporated into bilharziasis
- ❖ **What are the character of hepatitis in bilharzial patient with hepatitis?**
 - Worse than normal course due to:
 - ↑ incidence of carrier state
 - ↑ incidence of development to cirrhosis
 - ↑ incidence of development to malignancy
- ❖ **What are the values of PR in bilharziasis :**
 - Polyps, Piles, Prostate enlargement & Bladder malignancy
- ❖ **What is the main presentation of Intestinal Bilharziasis?**
 - Bleeding per rectum (Hematochezia).
- ❖ **When bilharziasis can cause bleeding per crectum?**
 - B polypi & Portal hypertension → secondary piles

Mixed bilharzial & hepatitis cirrhosis

(In this sheet, I will focus on differences between this case and the previous)

Personal history

❖ **Complaint:** Pain in left upper quadrant of abdomen of one week duration.

❖ HPI:

- The condition started 10 years ago by an attack of **hematemesis**, preceded by nausea and vomiting. The blood was dark red in colour, containing food particles, not associated with loss of consciousness, the patient was admitted to Zagazig university hospital where resuscitation was done by fluids and blood transfusion and endoscopy was done to stop bleeding, the attack was followed by melena for 3 days.
- The patient was followed up by regular sclerotherapy.
- The patient remained symptom free till 5 years ago when he experienced gradual onset, progressive course of

abdominal distension associated with bilateral pitting painless **lower limb oedema** reaching the mid leg level for which the patient was admitted to hospital, investigated by LFTs and abdominal ultrasonography and treated by diuretics.

- The patient remained symptom free till one week ago when he experienced gradual onset, progressive course of **localized dull aching pain** at Lt upper quadrant of the abdomen not related to position, respiration, exercise or meal.
- No history of abdominal swellings, other upper or lower GIT symptoms, hepatobiliary or urogenital symptoms, no constitutional symptoms.
- No symptoms of other system affection

❖ Past history

❖ Family history ...

1. General exam

A. Overview:

- **Temperature** : 37
- **Blood pressure** 110/70
- **Pulse** : regular pulse, 75 /minute, average volume, no special characters, vessel wall is not felt, equal on both sides with intact peripheral pulsations, no radiofemoral delay.
- The patient is **fully conscious**, well oriented for time place and persons, average mood and memory, the patient is co-operative with average intelligence, the patient looks well, average built, no jaundice, no pallor or cyanosis.

B. H&N

- No wasting of muscles. No parotid enlargement, no fine silky hair, no fetor hepaticus, the skin shows no spider naevi or paper money appearance
- Neck veins are pulsating not congested.

C. Extremities

- Mild bilateral pitting **L.L oedema** reaching mid leg level and not tender.
- No Wasting of muscles.
- No clubbing, no leuconychia, no capillary pulsations and no flappy tremors.
- The skin shows no spider naevi, no palmer erythema, no paper money appearance, no S.C haemorrhage.

2. Local exam :

A. Inspection

- The abdomen moves freely with respiration, generalized abdominal distension, there is no localized bulge, no dilated veins, regarding the skin: there is no scars, no stria no scratch marks, no sc haemorrhage, no pigmentation, no oedema in anterior abdominal wall with normal skin elasticity, there is no inguinal, femoral or umbilical hernias and no gynecomastia. There is wide subcostal angle confirmed by thumb test, no epigastric pulsation, no visible peristalsis. There is divarication of recti. Regarding the umbilicus, it is shifted downwards but inverted and transverse in shape no hernia no discharge. The skin shows no dilated veins no nodules no scars, pigmentation or ulcers, there is male distribution of suprapubic hair with normal genitalia, regarding the back no deformity, no swelling, no scars.

B. Palpation

❖ By superficial palpation:

- No swellings, no rigidity, no tenderness, no hyperesthesia.

❖ By deep palpation:

- The liver is firm in consistency, sharp border, Rt. lobe is felt 4 finger under Rt. costal margin in the right MCL, Lt. lobe felt 4 fingers below xiphisternum in midline, with smooth surface no tenderness, no pulsation.
- The spleen is firm in consistency, rounded notched border, it is felt 3 fingers below the left costal margin (it is huge in size) with smooth surface, it is not tender or pitted (to be differentiated from other masses in the left hypochondrium and left lumbar specially left kidney).
- Otherwise, there is no other palpable abdominal organs.

C. Percussion

- Upper border of liver is in the 5th intercostals space in the right midclavicular line detected by heavy percussion.
- There is dullness 4 fingers below Rt. costal margin and 4 fingers below xiphisternum continuous with hepatic dullness detected by light percussion.
- There is dullness detected 3 fingers below the left costal margin detected by light percussion and continuous with Traub's area dullness.
- There is no ascites detected by shifting dullness (search for mild ascites by percussion in knee elbow position).

D. Auscultation

- There is audible intestinal sound, no vascular sounds no hepatic or splenic rubs no succussion splash no puddle's sign. The liver is detected below the right costal margin by scratch test.

? What is your diagnosis?

❖ Diagnosis:

- A case of hepato-splenomegaly for D.D most probably mixed bilharzial cirrhosis, there is some signs of LCF and previous attacks of bleeding with no complications. Or
- A case of hepato-splenomegaly for D.D most probably mixed bilharzial cirrhosis, there is vascular and cellular decompensation with no complications

? Why ...?

- Splenomegaly? from local exam and sonar
- Mixed bilharzial cirrhosis? from history, urine and stool analysis, hepatitis markers and serological tests for bilharzia (rectal snip is much better)
- Cellular decompensation? from of symptoms and signs of LCF and LFTs
- Vascular decompensation? from signs of portal hypertension (splenomegaly, portal venography and manometry are much better) and bleeding.
- Not Complicated? as there is no evidence of spontaneous bacterial peritonitis, malignancy or hypersplenism (see diagnosis of abdominal case to know how to detect each one).

? How would you proceed to manage this case?

❖ Investigation

- Urine and stool analysis for viable bilharzial ova
- Blood for CBC (anemia), LFT, serological tests of bilharziasis, hepatitis markers
- Ascetic fluid analysis for
- ECG for bilharzial cor pulmonale
- Barium studies for
- Ultrasonography for
- Endoscopies upper and lower for

❖ Treatment

- Stop any hepatotoxic drugs
- Treatment of hematemesis in between attacks (band ligation better than sclerotherapy, TIPS can be tried)
- Treatment of ascites if present

HEMOLYTIC ANEMIA

- A. BETA Thalassemia "Thalassemia major, Mediterranean anemia"
B. Sickle cell anemia

History

- A. Abdominal sheet but focus on (pain, swelling, jaundice)
B. Cardiological sheet ($\downarrow$ COP, systemic congestion, pulmonary congestion)

1) **Personal history:** ...

2) **Common Complaint:** abdominal distention, pallor, yellowish discoloration of the eye, pain

3) **History of present illness:**

• Frequency of blood transfusion

• Anemia: symptoms & complications

- Pallor, Palpitation
- Dyspnea
- Easy fatigue, lassitude
- Headache, blurring of vision
- Failure to thrive; stunted growth



• Complications of repeated blood transfusion

❖ **Specific for Thalassemia major:**

• Onset (after 6 months)

• Positive family history

• Residence (Mediterranean)

• Complications of hemosiderosis: cardiomyopathy, HF, DM, cirrhosis (LCF), ... etc

• Operation for Splenectomy or cholecystectomy

• Complications of hypersplenism: bleeding tendency & increase the rate of blood transfusion

• Hemolytic crisis: fever, rigors, bone pains, abdominal pain, Lion pain, dark urine + anuria & shock

❖ **Specific for Sickle cell anemia**

• History of infection

• Autosplenectomy

• Abdominal pain

• Pain in hands & abdomen "hand & foot syndrome"

4) **Past history:** drugs causing hemolytic anemia

5) **Family history** of similar condition or consanguinity

Examination

❖ **General + local (abdominal examination):**

General Features of Hemolytic Anemia

1. **Signs of anemia:**

A. **Pallor:** best detected in mm of mouth, palmer creases & nail folds

B. **Hyperdynamic circulation:**

• **Pulse:** tachycardia, big pulse volume, water hammer pulse, exaggerated carotid pulsation.

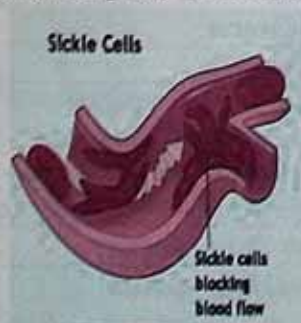
• **Heart:** Accentuated heart sounds, S3, haemic murmur

• **Haemic murmur should be differentiated from AR:**

• **Haemic murmur:**

- Heard all over the heart & best on the base of the heart (pulmonary area)
- Low intensity, soft systolic with no thrill

• In severe cases high COP HF



C. Oedema of LL may occur due to :

- Hypoxia which increases the capillary permeability
 - Salt & water retention
 - HF from chronic Anemic or cardiomyopathy
2. Failure to thrive : stunted growth , short stature
3. Spooning of nail due to:
- Iron deficiency anemia
 - Liver disease (hemochromatosis)

4. Hemolytic jaundice :

- A. Skin pigmentation : greenish brown (Pallor + Jaundice + hemosiderosis)
- B. Eye : mild jaundice : lemon yellow Jaundice
- C. Stool : dark (excess stercobilinogen)
- D. Urine is normal , but darkens if:
- Left to stand
 - Hemolytic crisis
 - Pigment stones
- E. Gall bladder : pigment stones (Scar for cholecystectomy)
- F. Chronic leg ulcers surrounded with pigmentation, oedema , ischemia
- G. Hepatomegaly



II. Specific Features in thalassemia major:

- Splenomegaly
- Scar for splenectomy
- Hemosiderosis : cardiomyopathy, HF, DM, cirrhosis,... etc
- Expansion of BM : Mongoloid face's :
 - Expansion of forehead "protrusion of frontal & parietal bone"
 - Depressed nasal bridges
 - Prominent maxilla
 - Protruding upper central incisors
- Crisis : 3 main types in thalassemia major :

	A. Hemolytic crisis	B. Aplastic crisis	C. Megaloblastic crisis
• Cause	◦ Sudden hemolysis with excess Hb	◦ Sudden depression of BM (viral infection)	◦ Folic acid deficiency (gradual)
❖ Clinical picture			
• Anemia	◦ Become worse	◦ Become worse	◦ Mild change
• Jaundice	◦ Become worse	◦ Constant	◦ Mild change
• Others	◦ Fever, rigors ,bone pains, abdominal pain ,Lion pain, dark urine + anuria & shock	◦ Symptoms of infection (pancytopenia)	◦ Symptoms of infection (pancytopenia)
❖ Investigations			
• Reticulocytes	◦ Increase	◦ Absent	◦ Absent
• BM	◦ Erythroid hyperplasia	◦ Absent erythroid series	◦ Megaloblastic changes

III. Specific Feature in sickle cell anemia:

- Autospplenectomy: from multiple splenic infarction
 - Palpation of spleen in adults with hemolytic anemia : never sickle cell anemia
- Vaso-occlusive crisis in sickle cell anemia only : painful occlusion of the blood vessels :
 - A. Infections : Chest, Bone (osteomyelitis)
 - B. Cardiac : MI
 - C. Chest : cor-pulmonale
 - D. Abdomen : Mesenteric occlusion; acute abdomen, Priapism
 - E. Neurology: Stroke: hemiplegia, Retinal detachment & blindness
 - F. Limbs : Leg ulcers, Avascular necrosis of femur, Hand & foot syndrome (dactylitis ممكن تلاقي الصواب مع طول بعض)

Shorter than peers

Crises:
hours-weeks long
pain & swelling
in
Chest
Abdomen
Hands & feet



Discussion :

❖ What are the causes of severe pallor in this case?	❖ What are the causes of deepening of jaundice in this case?
❖ When is anemia marked in hemolytic anemia?	❖ When is jaundice marked in hemolytic anemia?
• Hemolytic crisis • Aplastic anemia • Megaloblastic anemia • Hypersplenism	• Hemolytic crisis • Obstructive jaundice • LCF • Hepatitis • Incompatible blood transfusion

Diagnosis :

- ❖ A case of jaundice for DD most probably hemolytic anemia most probably thalassemia major or sickle cell anemia complicated by hemosiderosis, liver cirrhosis & hypersplenism
- ❖ **Why hemolytic anemia :**
 - History : repeated blood transfusion
 - Examination : pallor, jaundice, hepatosplenomegaly
 - Investigation : ↑ reticulocytic count

Why :

	A. Thalassemia major	B. Sickle cell anemia
• Common in	○ Mediterranean countries	○ Negros
• By history	○ Residence	○ History of vasoocclusive syndrome
	○ Positive family history	○ History of infection
• By examination	○ Face "thalassemic facies"	○ Evidence of vasoocclusive e.g. stroke
	○ Huge spleen	○ Impalpable spleen
• By investigation	○ Type of anemia : hypochromic microcytic	○ Sickle cell
	○ Hb electrophoresis (HbF)	○ Hb electrophoresis (HbS)

HUGE SPLENOMEGALY

- ❖ **Definition :** enlarged spleen that crosses the midline
- ❖ **Etiology :** see before in the abdomen
- ❖ **History & examination :**
 - As abdomen but focus on the following to know the diagnosis of the case:



1. Thalassemia major: see before
 - A. Age of onset : childhood
 - B. Symptoms & signs of anemia symptoms & signs of hemolytic anemia
 - C. Symptoms & signs specific to thalassemia major

3. Portal hypertension: 2ry to:
 - A. Post hepatitis cirrhosis:
 - History of blood transfusion
 - Signs of LCF
 - B. Bilharziasis:
 - History of B, contact with canal water

5. Amyloidosis
 - History of other system affection e.g. renal or chest (SLS)

2. Chronic myeloid leukemia:
 - A. Symptoms & signs of anemia
 - B. Prolonged fever & recurrent infection
 - C. Bleeding tendency for gum, epistaxis, skin for purpura ecchymotic patches
 - D. Bony pain (arthralgia, arthritis) & sternal tenderness
 - E. History of sternal puncture
 - F. Other swellings in the body: Examination of all LN of the body
 - G. Gum hypertrophy

4. Malaria & kala azar
 - History of travelling to other countries
 - History of fever & rigors

BLEEDING TENDENCY

❖ Complaint :

- Reddish spots with or without mucous membrane bleeding
- Post – traumatic swelling

❖ History & examination : as abdomen but focus in the following to know the diagnosis of the case :

❖ Types	A. Purpura (In vascular / platelet defect)	B. In coagulopathy e.g. Hemophilia
❖ Age	○ Any age	○ Since birth e.g. Circumcision ○ After operations : Tooth extraction , tonsillectomy
❖ Sex	○ Females	○ Males
❖ Family history	○ Negative	○ Positive
❖ Hess test (capillary fragility test)	○ Positive	○ Negative

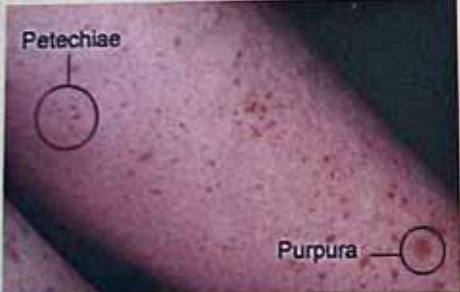
- Hess test : Method :
 - A circle 5 cm in diameter is marked on the antecubital fossa.
 - The sphygmomanometer is inflated to midway between SBP & DBP for 5 minutes.
 - Count the number of petichae within the circle
- Result : positive test means the presence of more than 10 petichae
- Value : it is positive in all cases of purpura



❖ Characters:		
❖ Site of bleeding :		
▪ Cutaneous	○ Petichae , purpura , ecchymosis	○ Ecchymosis only
▪ Orificial	○ Epistaxis, hemoptysis, hematemesis , Bleeding per rectum & melena , hematuria.	
▪ Mucous membrane	○ Oral, gingival , conjunctival bleeding .	
▪ Internal	○ Intracranial hemorrhage (convulsion , ↑ ICT symptoms) , retinal hemorrhage, cavitory hemorrhage (serous membranes; pleura, pericardium, peritoneal hemorrhage)	○ Hemoarthrosis: spontanous bleeding in joints (pain, swelling & movement limitation) → fibrosis & deformity ○ Muscle hematoma after IM injection, others like purpura
❖ Mechanism of bleeding	○ Spontaneously or post-traumatic	○ Spontaneously or post-traumatic
❖ Character of bleeding	○ Immediate & of short duration < 1 day	○ Delayed: days, weeks, months
❖ Severity	○ Mild bleeding (ooze)	○ Severe = profuse

☠ Remember :

- Reddish blue or purple discoloration of the skin that doesn't blanch on pressure :

○ Petechiae : less than 2 mm	○ Purpura: 2 mm to 1 cm	○ Ecchymosis : more than 1 cm
		

Petechiae	Ecchymosis
○ Intradermal hemorrhage	○ Subcutaneous hemorrhage
○ mm	○ cm
○ Not raised	○ Raised
○ Disappears without color changes	○ Color changes
○ Not related to the site of trauma	○ Usually related to the site of trauma

Specific characters of the cause:

Liver failure: LCF symptoms & sign

Aplastic anemia:

- History of : Repeated blood transfusion, Cytotoxic drug intake, Prolonged course
- Similar condition in the family : Fanconi syndrome
- Skin examination: Purpuric eruption Generalized, variable size, not raised above the surface of the skin & doesn't blanch on the pressure

Immune Thrombocytopenic Purpura (ITP):

A. ITP in child

- Sudden acute onset with regressive course & short duration
- Equal sex

History of preceding fever (viral infection)

General features of purpura : see before

Skin examination: Purpuric eruption: Generalized, variable size, not raised above the surface of the skin & doesn't blanch on the pressure

Spleen is not palpable, if palpable never hugely enlarged

No blood transfusion, if there is blood transfusion, it's usually once

B. ITP in adults

- Chronic gradual onset with remission & relapses course

- Female more than males

- Associated with other autoimmune disorders e.g. SLE

Henoch - Schonlein purpura:

History of : Arthritis, Abdominal pain, GIT bleeding, Hematuria

Skin examination: raised palpable purpuric eruption (rash) with itching (vasculitis) especially over the buttocks & extensors surface of legs

Malignancy e.g. Leukemia:

History of progressive course of Prolonged continuous fever, Bony pain & Recent marked weight loss

Examination: Abdominal distension (HSM) & Any other swellings (LNs), bony pain

Hemophilia:

History of bleeding Since birth e.g. Circumcision & After operations : Tooth extraction, tonsillectomy

Muscle hematoma after IM injection

Hemoarthrosis: joint pain, swelling (knee effusion), movement limitation → fibrosis & deformity

Examination :

❖ فحص كامل لأن السبب ممكن يكون systemic مع التركيز على :

I. General examination:

- Skin for purpuric eruption & it's character
- Eye for conjunctival hemorrhage
- Mouth : gum bleeding
- Pallor in aplastic anemia (usually no pallor with ITP)
- Upper limb : Hess test
- Lower limb : knee effusion (hemoarthrosis); if +ve → coagulopathy

II. Systemic examination:

- Cardiac : hemorrhagic pericardial effusion
- Chest : hemorrhagic pleural effusion
- Abdominal :
 - Manifestations of liver cell failure
 - LN examinations to exclude leukemia
 - Spleen : hypersplenism & leukemia
 - Hemorrhagic ascites
- CNS: to exclude hemorrhage "cerebral or subarachnoid" : focal deficit "
- Joint : hemoarthrosis & Bone tenderness with leukemia

Diagnosis

- A case of generalized purpuric eruption without pallor without organomegaly without lymphadenopathy most probably ITP post viral infection.
- A case of inherited coagulation defect most probably hemophilia



GENERALIZED OEDEMA

❖ Causes	A. Right side heart failure (all causes)	B. Liver cell failure	C. Nutritional deficiency	D. Angioneurotic	E. Renal causes
❖ History as abdomen:	Cardiac history especially right side manifestation	LCF Symptoms	Nutritional habits	History of drug intake (allergy)	Renal symptoms
❖ Examination: ❖ General & Local (abdominal)	- Character of cardiac oedema : see cardiology. - Oedema of LL always precedes appearance of ascites except in 2 conditions : A. Pericardial effusion & constrictive pericarditis B. TR - Hypotension in HF - Congested Pulsating neck veins . - Chest : infection, effusion : very important (pneumonia)	- Ascites occurs first then LL oedema - It's associated with features of liver disease - Examine : Abdomen : liver, jaundice, spleen ascites	- It occurs in severe Nutritional deficiency - Associated with other features of nutritional deficiencies	- This allergic oedema occurs with constant relation to certain factors e.g. eating certain food or inhaling certain substances - Acute onset especially lids, lips, larynx, but may be generalized - There is usually +ve family history of oedema or other forms of allergy - The patient himself may have other forms of allergy - There is characteristic rapid response to anti-allergic measures	- Oedema occurs first in eye lids & is associated with features of renal disease A. Nephritic : acute onset of oliguria , mild hematuria, (smoky urine), minimal edema, hypertension B. Nephrotic : edema , hyperlipidemia

RENAL OEDEMA

❖ **History** : as abdominal sheet but focus on the following points :

• Personal history : ...

• Complaint : puffiness of eyelids

• Present history :

I. **Analysis of the complaint**; OCD, $\uparrow\downarrow$, associated symptoms

II. **Symptoms of the same system** :

1. Urinary symptoms : change in the amount or in colour

○ Oliguria & hematuria in nephritic syndrome

2. Headache (hypertension) : in nephritic syndrome

3. Symptoms of complications :

▪ Chest infection : fever, dyspnea

▪ Skin infection

▪ Urinary tract infections: dysuria, ... etc.

▪ Abdominal : pain ,vomiting (peritonitis or GIT congestion) & diarrhea

▪ Rash , arthritis (with lupus nephritis – Henoch Schonlein purpura)

4. Investigations & treatment :

• Steroids or immunosuppressive & hospitalization

III. **Symptoms of other systems** :

• Hepatic & cardiac to exclude other causes of edema

❖ **Examination**: as general & local abdomen but focus on :

I. **General examination**:

• BP: normal in nephrotic & high in nephritic ,

❖ **N.B.:** causes of hypertension in Nephrotic syndrome :

○ Nephritic nephrotic

○ Collagen diseases e.g. SLE

○ Complication of atherosclerosis or renal failure

○ Steroids

• Puffy eyelids

• Oedema start periorbital then face then sacrum then genitalia then general

• Pallor

• Xanthomata (hyperlipidemia)

• Cushinoid features in long standing treatment with steroids

• Lower limb edema : bilateral pitting not tender reaching ...

II. **Abdominal examination**:

• Generalized distention of the abdomen

• Stretched skin, Visible veins

• Divarication of recti

• Wide subcostal angle

• Tender & rigid abdomen suspect in peritonitis

• Ascites examination

• Scrotal edema

❖ **Diagnosis** :

• A case of generalized pitting oedema (associated with moderate or tense ascites for investigation) most probably nephrotic syndrome in (first attack or in relapse) complicated by acute bronchitis

❖ **Why nephrotic ?**

○ Character & distribution of edema

○ Edema started over eye "periorbital"

○ Gradual onset

○ Absent hematuria $\pm$ normal blood pressure.



Chronic Hemolytic Anemia

❖ Personal history : ...

❖ **Complaint** : Pain in Lt. upper quadrant of abdomen of 2 weeks duration.

❖ HPI:

- The condition started since he was 12 years old by gradual onset, progressive course of **localized dull aching pain** at Lt upper quadrant of the abdomen not related to position, respiration, exercise or meal. The condition was associated with **fever, chills, bony pain, headache**, with **jaundice** of acute onset, progressive course with **dark urine** with no anorexia, nausea, vomiting, itching, cachexia, or fatty dyspepsia. Then the patient was admitted to hospital and investigated by liver function test which was normal and CBC which revealed severe anemia and the patient was advised to take regular Bl. Transfusion every 3 months.
- At age of 25 y the patient re-experienced the same attack of **fever, chills, headache, bone pain, jaundice** of acute onset, progressive course, **dark urine**, no anorexia, nausea, vomiting, cachexia or fatty dyspepsia. he was admitted to hospital and advised to take blood transfusion every month with some medication as folate, desferal.
- The patient remained quiet well until 2 weeks ago when he re-suffered from dull aching **pain** at Lt. upper quadrant of abdomen of gradual onset, progressive course, not related to position, exercise, meal, respiration
- Apart from regular exertional **palpitation**, there is no symptoms suggesting cardiovascular system affection.
- No history of other system affection

❖ Past history:

- No history of operations, drug known to cause haemolysis, common diseases (DM, HPN).

❖ **Family history** : +Ve consanguinity, No Similar condition in family, No common diseases in family.

❖ General examination :

- **Temperature**: 37° c., **Bl. Pressure**: 140/60.
- **Pulse**: 70 beat/minute, big pulse volume, equal in both side, with water hammer pulse, blood vessels not felt, intact peripheral pulsation with no radio-femoral delay.
- **Mentality**: The patient is fully conscious, well oriented for time, place and person. Average mood and memory. The patient is co-operative with average intelligence.
- **Built**: Average Built, **Head & Neck**: pallor, jaundice, mongoloid features.
- **UL**: there is pallor in palmar creases. (now improved)
- **LL**: there is pigmentation (may be ulcers specially in sickle cell anemia).

❖ Local examination :

✚ **By Inspection**: There is local swelling in Lt. upper quadrant of abdomen decreased by rising up test.

✚ Palpation:

- **By superficial palpation**: There is swelling in lt hypochondrium. No other swellings, rigidity or tenderness.
- **By deep palpation**:
 - The liver is firm in consistency, sharp border, Rt. lobe is felt 2 finger under Rt. costal margin in the right MCL, lt. lobe felt 3 fingers below xiphisternum in midline, with smooth surface no tenderness, no pulsation.
 - The spleen is firm in consistency, rounded border, huge in size, smooth surface, not tender, not pitted no other palpable abd. Organs.

✚ Percussion:

- Upper border of liver is in Rt. 6th intercostal space by heavy percussion with hepatic span is 16 cm at right MCL.
- There is dullness below Rt. costal margin, xiphisternum continuous with hepatic dullness detected by light percussion.
- There is dullness exceeding umbilicus and continuous with traub's area dullness detected by light percussion.

✚ **By Auscultation of the heart**: There is accentuated 1st and 2nd heart sounds, soft systolic murmurs all over pericordium (hemic murmurs) .

❖ Investigations :

- HB electrophoresis: detect fetal Hb (HbF -->80-90%)
- CBC: hypochromic microcytic anemia, reticulocytosis.
- Target cell in blood film.

❖ **Treatment** : Avoid precipitating factor (if possible), Repeated Bl. Transfusion, Desferral, Folic acid.

❖ **Diagnosis** : A case of hepato-splenomegaly for D.D most probably Chronic Hemolytic anemia most probably thalassemia major

LYMPH NODES

❖ Head & neck:

I. Circular group:

- A. Inner ring : Waldeyer's ring: two palatine tonsil, lingual tonsil & pharyngeal tonsil
 B. Outer ring:

1. Submental L.N.s	2. Submandibular L.N.s	3. Facial L.N.s	4. Parotid L.N.s	5. Pre-auricular L.N.s	6. Post-auricular L.N.s	7. Occipital L.N.s
❖ Site:						
○ In submental Δ	○ In sub-mandibular Δ	○ On buccinator muscle	○ In parotid substance	○ In front of tragus	○ On the mastoid process	○ Between mastoid process & external occipital protuberance
❖ Afferent (drains):						
○ Central part of tongue ○ Floor of mouth. ○ Middle part of lower lip	○ Inner angle of eye & side of nose. ○ Cheek, angle of mouth, upper lip, side of tongue & lower lip except middle part	○ Part of cheek	○ Front of scalp	○ Side of scalp	○ Temporal part of scalp	○ Back of scalp
❖ Efferent:						
○ Submandibular L.N.s but few Lymphatics into jugulo-omohyoid L.N.s → Lower deep cervical L.N.s	○ Jugulo-omohyoid group of L.N.s → Lower deep cervical L.N.s	○ Upper deep cervical L.N.s.	○ Upper deep cervical L.N.	○ Upper deep cervical L.N.s	○ Upper deep cervical L.N.s	○ Lower deep cervical L.N.s

II. Vertical group:

1. Superficial group:

- Superficial lateral cervical lymph nodes along the external jugular vein
- Superficial anterior cervical lymph node along the anterior jugular vein

2. Deep group:

A. Middle line L.N.s (Median Group) :

- Pre-laryngeal L.N.s (Delphian LN) located anterior to the thyroid cartilage
- Pre-tracheal L.N.s located anterior to the trachea
- Supra-sternal L.N.s. on supra-sternal notch

B. Lateral group: deep cervical L.N.s:

- Deep cervical L.N.s which lie deep to sternomastoid muscle
- Divided into Upper deep cervical L.N.s & Lower deep cervical L.N.s by intermediate tendon of omohyoid muscle
- Divided into anterior & posterior by internal jugular vein
- N.B.: Jugulo-digastric L.N.: is the first L.N. in the deep cervical & is located at the angle of the mandible

❖ **Breast & trunk:**

- Palpate axillary & supra-clavicular L.N.s:

A. Axillary L.N.s: They drain the upper limb down to umbilicus. They arranged in 5 groups:

1. The (Anterior) Pectoral group	2. The (posterior) Sub-scapular Group	3. The (Lateral) Humeral Group	4. The (medial) Central Group	5. The Apical Group
❖ Site				
○ Under cover the pectoralis major.	○ Along posterior axillary fold	○ Along upper part of humerus	○ Central part of axilla	○ External apex of axilla
❖ Drains				
○ Chest wall. ○ Whole breast except tail. ○ Anterior abdominal wall above umbilicus	○ Axillary tail ○ posterior abdominal wall above umbilicus	○ All the upper limb.	○ 1 + 2 + 3	○ 1 + 2 + 3 + 4 + infra-clavicular L.Ns.

B. Supra-clavicular L.N.s:

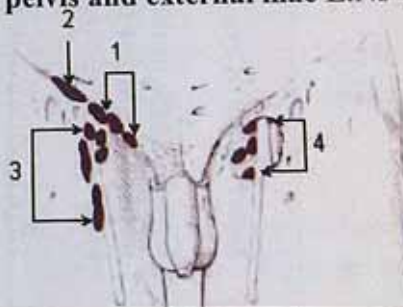
- Site : above clavicle.
- Right Supra-clavicular L.N. is called Scalene L.N. while left Supra-clavicular L.N. is called Virchow L.N.
- Drains : from internal mammary L.Ns

❖ **Upper limb :**

A. Superficial group of L.Ns:	B. Deep group of L.Ns:
○ Supra-trochlear (Epitrochlear) group of L.Ns	○ Lateral (Humeral) group of L.Ns
❖ Site	
○ Above medial epicondyle of humerus	○ At surgical neck of humerus
❖ Afferent	
○ Same as delto-pectoral group.	○ Drains all upper limb (deeply).
❖ Efferent	
○ Deep group of L.Ns	○ Apical group of axillary L.Ns.

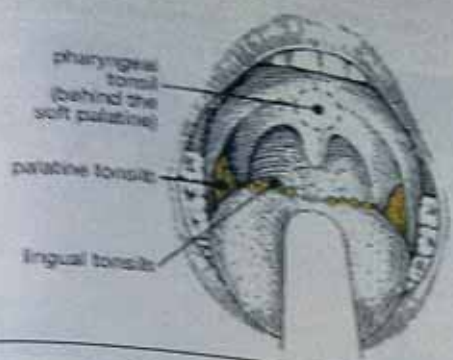
❖ **Abdomen :**

- A. External & Internal iliac L.Ns : along correspond arteries from umbilicus to mid-inguinat point
- B. Common iliac L.Ns : along correspond arteries from umbilicus to mid-inguinat point
- C. Para-aortic group of L.Ns
- Site: One on each side of aorta from xiphisternum to umbilicus
 - Afferent : Drains internal Iliac L.Ns which drain pelvis and external iliac L.Ns which drain deep inguinal L.Ns
 - Efferent : Cysterna chyli

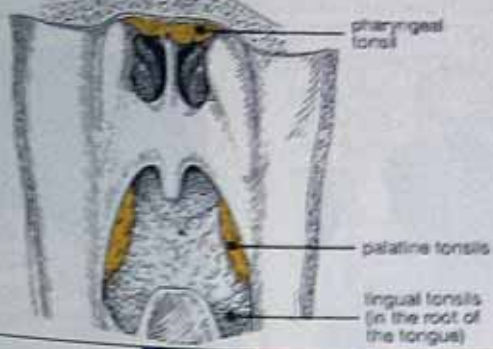
❖ **Lower limb:**

I. Superficial groups of L.N.s		II. Deep groups of L.N.s (4)
A. Vertical limb : lateral to long saphenous vein (3)		○ Along the femoral vein ○ The largest called Cloquet
B. Transverse limb : below & parallel to inguinal ligament: 2 parts		
♂ Medial part (1)	♂ Lateral part (2)	
❖ Drains:		
○ Anterior abdominal wall below level of umbilicus.	○ Post abdominal wall below level of umbilicus.	○ Glans penis ○ All lower limb
○ The perineum	○ The gluteal region.	
○ The skin of external genitalia except glans penis.		

FROM THE FRONT
-anterior view



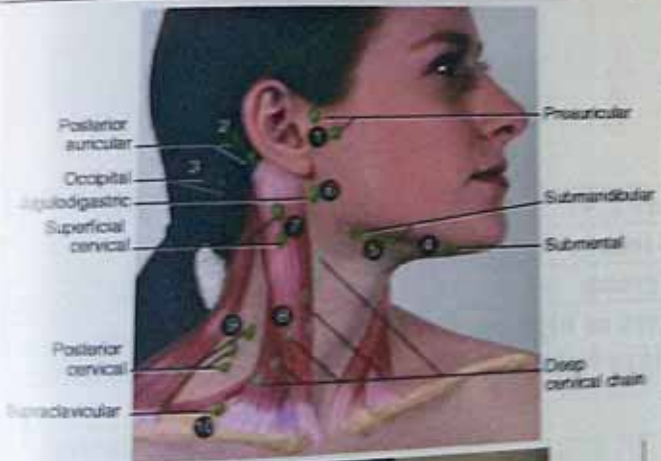
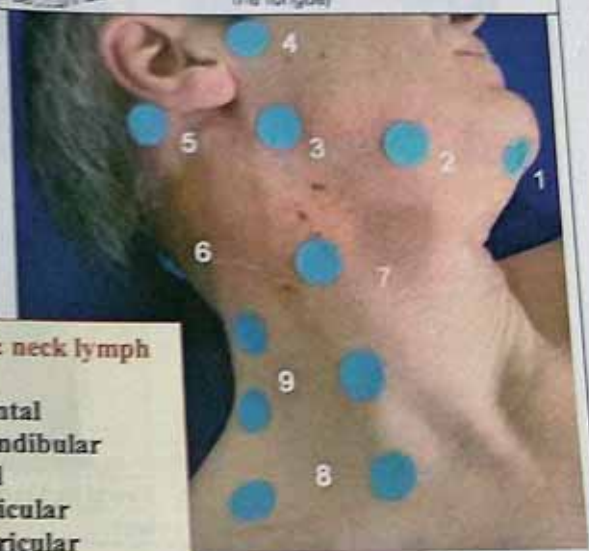
FROM THE BACK
-posterior view



- | | |
|------------------------|--------------|
| Superior deep cervical | Pretracheal |
| Inferior deep cervical | Paratracheal |
| Submental | |
| Submandibular | |

❖ Head & neck lymph nodes:

1. Submental
2. Submandibular
3. Parotid
4. Preauricular
5. Postauricular
6. Occipital
7. Anterior Cervical
8. Supraclavicular
9. Posterior Cervical

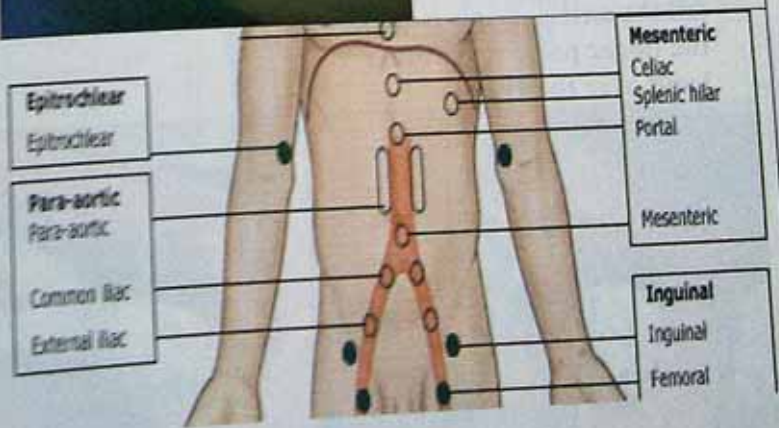


Epitrochlear lymph node



❖ Axillary lymph nodes

1. Central
2. Humeral
3. Pectoral
4. Apical
5. Subscapular



- Epitrochlear
Epitrochlear
- Para-aortic
Para-aortic
- Common iliac
External iliac

- Mesenteric
Celiac
Splenic hilar
Portal
- Mesenteric

- Inguinal
Inguinal
Femoral

❖ Examination of lymph node: Technique for palpation:

❖ Head & neck:

↳ From front : Facing the patient :

○ Occipital LN



○ Post-auricular L.N.



○ Posterior cervical



↳ From behind of the patient :

○ Pre-auricular LN



○ Submental LN



○ Submandibular: tilt the head to the side you examine



↳ From behind of the patient & head slightly flexed

○ Upper deep cervical



○ Lower deep cervical



❖ Axillary LN:

↳ From front :

A. Anterior (pectoral) group

- Move your hand anteriorly behind the pectoralis major muscle down to axillary tail to feel the anterior group against the finger pads of the other hand over the chest



B. Apical group

- Push the tips of fingers as high up into axilla as possible to feel the apical group



C. Medial (central) group

- By downward sliding movement



From side :

D. Lateral (humeral) group
Stand by the patient's side, put the fingers of both hands to deep aspect of upper end of the humerus to palpate the lateral axillary wall while thumbs rest on the deltoid region



E. Posterior (subscapular) group
Stand behind the patient

Palpate the posterior fold of axilla with palm looking backwards, the posterior group is palpated against the thumb of the same hand



❖ Other technique :



From behind :

F. Supra-clavicular L.N.s

Stand behind the patient and press 3 fingers of each hand into corresponding supraclavicular fossa then ask the patient to elevate the shoulder upwards and forwards then roll your fingers to feel the L.N.



❖ Upper limb : epitrochlear L.N.: with elbow flexed 30°, palpate in the groove between biceps & triceps about 3cm above medial epicondyle



❖ Abdomen : para-aortic L.N. by Rolling technique



❖ Lower limb

Vertical limb of superficial group



Transverse limb of superficial group



LYMPHADENOPATHY

Introduction:

Generalized Lymphadenopathy:

- ❖ Starts as one group then becomes generalized:
 1. T.B. (2ry) rare : Night sweat & night fever + loss of weight & loss of appetite
 2. Lymphoma (Hodgkin & non Hodgkin): multiple swelling at anatomical site of L.Ns
- ❖ Starts generalized from the start:
 1. Leukemia: Bone ache + bleeding tendency from orifices:

• Mouth: ○ Bleeding gums. ○ Hemoptysis. ○ Hematemesis	• Nose: Epistaxis • Rectum :Bleeding • Bladder Hematuria • Vagina: Menorrhagia
--	---
 2. 2ry syphilis: Skin rashes + genital ulcer
 3. Infectious mononucleosis (IMN): Skin rashes + glandular fever
 4. AIDS

Localized Lymphadenopathy:

1. T.B.(1ry) : No toxemia : Young + bad hygiene + cold abscess
2. Acute lymphadenitis :1ry septic or malignant focus e.g. ulcer or swelling or septic trauma at draining sites

Remember

	1ry TB	2ry TB
▪ Incidence	○ Common	○ Rare
▪ Route	○ Lymph borne	○ Blood borne
▪ Clinical picture	○ Localized lymphadenopathy (Upper deep cervical L.Ns) ○ No toxemia. ○ L.Ns : - Painless, enlarged. - (Firm, cystic, hard) - Matted or rosary beads (Can be counted)	○ Generalized lymphadenopathy (Lymphadenoid.) ○ Toxemia. ○ L.Ns : - Painless, enlarged. - Firm. - Discrete & mobile (uniform size)
▪ Complications	○ Cold abscess ○ Calcification ○ T B sinus	-----

Acute lymphadenitis	Non Hodgkin lymphoma	Hodgkin lymphoma (Lymphadenoma)
○ Tender & enlarged	○ Painless & enlarged	○ Painless & enlarged
○ Firm	○ Firm, Soft, Hard	○ Firm
○ Single	○ Amalgamated (can't be counted)	○ Discrete & mobile (different size)
---	○ Infiltration (bad prognosis)	○ No infiltration but pressure symptoms.

Don't forget:

- Lymphadenoid = 2ry T.B
- Lymphadenoma = Hodgkin's disease
- Adenolymphoma = Adenocystic lymphoma (Warthin's tumor) of salivary gland.

LYMPHADENOPATHY HISTORY

Personal history:

1. Name: الاسم ثلاثي		2. Age: ☐ Young : TB ☐ Adult : Acute leukemia or Hodgkin (10-30 years). ☐ Old : Chronic leukemia or Non Hodgkin (30 - 70 years)	3. Sex : ☐ Malignancy more common in male.
4. Occupation: ☐ Brucellosis (in those contact with animal) ☐ Because brucellosis → Pel Epstein fever which is similar to Hodgkin's disease.		5. Marriage. 6. Menstrual history if female.	7. Residence & address: T.B (low socioeconomic standard area)
8. Habits of medical importance: ☐ Alcohol : induces pain at site of Hodgkin's disease		9. Handedness.	

Complaint + duration: ؟ أي التي جانبك المستشفى

- Multiple swellings (at anatomical site of L.Ns) ± pain

History of present illness = HPI ؟ آخر مرة كنت كويين فيها امتي ؟

I. Analysis of the complaint : swelling ± pain

- A. Analysis : OCD, site & side if localized , number if multiple ask about one group, associated other swelling if generalized
- B. Pain if present; analyze :
- Causes of painful LN:
 - Acute lymphadenitis
 - Late lymphoma
 - OCD, ↑ (e.g. alcohol) , ↓, Associated symptoms
 - Character
 - Site
 - Radiation
 - Relation to (meal, respiration, exertion, postural)

III. Systemic symptoms (general complications)

- A. Toxic manifestations (FHMA):
- Hectic fever : As in acute lymphadenitis (abscess).
 - Night fever : As in T.B. (2ry).
 - Glandular fever: [Fever + rashes] as in I.M.N.
 - Pel Epstein fever = Irregular = periodic: As in Hodgkin
- B. Etiological manifestations (see introduction)
- Generalized lymphadenopathy
 - Localized lymphadenopathy

IV. Investigations (Biopsy) & Treatment received .

II. Pressure (infiltration) symptoms (local complications)

- In neck lymphadenopathy
 - Dyspnea (trachea or larynx).
 - Dysphagia (oesophagus).
 - Horner's syndrome (sympathetic chain).
 - Face oedema (int. jugular v. compression)
 - Fainting attacks (carotid a. compression)
 - Hoarseness (recurrent laryngeal nerve).
- In chest lymphadenopathy: Chest pain, cough and dyspnea
- In axillary lymphadenopathy :
 - Oedema (Vein compression),
 - Ischemia or gangrene (Arterial compression)
 - Tingling, numbness... (Nerve compression)
- In abdominal lymphadenopathy:
 - Abdominal pain or back pain.
 - Jaundice (L.Ns in porta-hepatis).
 - Leg oedema (compressed iliac veins or I.V.C by iliac & Para-aortic lymph nodes).
 - Renal pain (ureteric compression)
- In inguinal lymphadenopathy: Same as axillary but ask about:
 - Varicose veins of L.L.
 - Claudication pain on walking

Fast history:

- Similar condition i.e. Recurrence
- Important disease as D.M., hypertension, heart diseases etc.
- Previous operation or biopsy (which L.Ns ?)
- The moderate size because {not big (degenerated) & Not small (no pathology)}
- Previous exposure to irradiation

Family history: T.B. may affect members (same environment)

I. General examination:

A. Vital signs

B. General examination :

- ill with cachexia as in late lymphoma
- Toxic face as in acute lymphadenitis.
- Under built as in T.B & lymphoma.

C. Systemic examination: Examine all accessible L.Ns except the presenting group (in local exam) + detection of the cause.

❖ Head	❖ Neck	❖ Upper & lower limb
○ Skull: for bone metastasis ○ Eye : for jaundice (if L.Ns in porta-hepatis) ○ Lip: for pallor & cyanosis (if L.Ns in mediastinum). ○ Tongue: Paralysis (If infiltration of hypoglossal nerve). ○ Parotid region for swelling : Mikulicz (auto-immune)	○ Thyroid gland: for enlargement, causes : A. toxic goiter (auto-immune) B. Hashimoto's thyroiditis (auto-immune) C. Occult carcinoma ○ Trachea : Central or not. ○ Carotid pulsation : cervical L.Ns ○ Congested neck veins : mediastinal L.Ns ○ Other L.Ns : If not the presenting group.	○ Venous oedema ○ Arterial pulsation ○ Nervous sensation ○ Other L.Ns if not the presenting group

❖ Chest examination	❖ Abdominal examination	❖ Neurological examination
○ Bone (chest wall) metastasis. ○ Lung (cavitation) as in T.B. ○ Sternum (tenderness) as in leukemia. ○ Despine's sign: mediastinal L.Ns (see chest)	○ H.S.M as in leukemia. ○ Abdominal organs as in spleen. ○ L.Ns if not the presenting group. ○ Testis : if testicular tumors ○ N.B.: Seminoma one of occult carcinoma ○ PR or PV = for pelvic tumors or nodule in the doglus pouch ○ DON'T FORGET (BACK): For metastasis	○ TB: Pott's disease ○ Luekima : infiltrate CNS: paraplegia & optic nerve affection ○ Lymphoma: paraplegia & peripheral neuritis

II. Local examination:

I. Inspection:

1. Number: Single or multiple i.e. localized or generalized

2. 8 S:

• Site (Anatomical site of L.Ns):

○ T.B.: Upper Deep Cervical L.Ns.	○ Hodgkin: Lower Deep Cervical L.Ns.	○ 2ry syphilis: Epitrochlear L.Ns.	○ IMN: Occipital L.Ns
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- Side : Rt. or Lt or both
- Shape : Oval, rounded or irregular
- Size : in ... cm x ... cm
- Surface : Smooth, nodular or lobulated
- Skin over :



○ Redness : If acute lymphadenitis.	○ Infiltration : If lymphoma.	○ Sinus : If T.B. or cold abscess.
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• Special sign:

○ Transmitted pulsation : If para-aortic L.Ns.	○ Moving up & down : If pre-tracheal L.N
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• Other swellings:

- If generalized lymphadenopathy look for other L.Ns all over the body.
- If localized lymphadenopathy look for infectious or malignant focus at draining area.

- In cervical lymphadenopathy : Examine (oral cavity).
(Tongue, teeth, cheek, lips, tonsil, thyroid, face, scalp, parotid, pharynx & larynx)
- In axillary lymphadenopathy : Examine breast, upper limbs, anterior wall of the trunk until level of umbilicus & posterior wall of the trunk until level of umbilicus
- In supra-clavicular L.Ns (Virchow's gland): above clavicle
- In inguinal L.Ns : Examine lower limbs, genitalia, perineum, anal canal, gluteal region & anterior abdominal wall below level of umbilicus.
- Edge : well or ill defined (or difficult to be seen)
- Distal effect: oedema, colour changes & trophic changes , deformity

II. Palpation: (technique for palpation : before)

1. Temperature : warm as in acute lymphadenitis
2. Tenderness : tender as in acute lymphadenitis
3. Mobility of LNs to each other:
- Discrete : 2ry TB & early Hodgkin
- Matted : 1ry TB, fused & can be counted
- Chain : TB
- Amalgamated : Non Hodgkin, fused & can't be counted
4. 8 S:
- Site, side, shape , size, surface : to confirm the inspection
- Skin over: To show if swelling attached to skin or not) by
- Sliding the skin or pushing mass under skin: If puckering = infiltrated = lymphoma.
- Special sign : to confirm the inspection
- Other swelling : as inspection
5. Edge : well or ill defined
6. Consistency :

○ Hard: Calcified 1ry T.B. or Non Hodgkin	○ Soft: Degenerated non Hodgkin	○ Cystic: Cold abscess	○ Firm: Acute lymphadenitis, 1ry T.B., 2ry T.B. & lymphoma
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7. Deep structure : Relation to deep muscle
8. Distal effect: oedema, colour changes & trophic changes , deformity

III. Percussion : sternum for mediastinal mass & tenderness as in leukemia

IV. Auscultation : Despine's sign (mediastinal LN) : see chest

❖ Discussion :

1. How can you examine 'matted' T.B L.Ns ?
 - Hold 2 adjacent L.Ns, one on each hand than move them in opposite directions → never separated.
2. What is the cause of 'Rosary beads' ?
 - Lymphangitis + lymphadenitis
3. What is the surgical importance of supraclavicular L.Ns ?
 - ↳ Lt. supra-clavicular L.Ns :
 - Below diaphragm (Cancer stomach, cancer colon & hypernephroma)
 - Above diaphragm (Lt. cancer breast & Lt. bronchial carcinoma)
 - ↳ Rt. Supra-clavicular L.Ns:
 - Below (Bare area of liver only).
 - Above (Rt. cancer breast only).
4. What are roles of 'Waldeyer's' ?
 - Waldeyer's ring.
 - Waldeyer's ligament.
 - Fascia of rectum.
 - Fascia of lower 1/3 ureter.
5. How can you DD between submandibular gland & L.Ns ?
 - Submandibular L.Ns only rolled on lower border of mandible.
6. How can you DD between parotid gland & L.Ns ?
 - It is very difficult so → Biopsy must be done.
7. What Is the value of occipital L.Ns enlargement ?
 - Enlarged with IMN (glandular fever)
 - N.B.: Don't Forget:
 - Spinal accessory L.Ns enlarged with pediculosis & frunculosis.
 - Jugulo-omohyoid L.Ns enlarged with Cancer tongue.
 - Jugulo-digastric L.Ns enlarged with tonsillar diseases "tonsillar L.Ns"
8. What is the value of epitrochlear L.Ns enlargement ?
 - Enlarged with 2ry syphilis.
9. Where are L.Ns arranged along [Arteries, veins & nerves] ?
 - L.Ns arranged along veins : [Limbs and head & neck]
 - L.Ns arranged along arteries : [Abdomen]
 - L.Ns arranged along nerves:
 - Epitrochlear L.Ns [ulnar nerve],
 - Accessory L.Ns [spinal accessory nerve]
 - L.Ns along [Lat. popliteal nerve]
10. Which LN enlargement means Generalized lymphadenopathy ?
 - Epi-trochlear LN (above medial epicondyle of humerus)
11. Which LN could be examined by stethoscope?
 - Interbronchial LN (D'spine sign in chest)

2ry T.B. sheet

❖ **Personal history:** ...

❖ **Complaint :** Multiple bilateral swellings in the neck, axilla & groin 2 years ago

❖ **HPI:**

- The condition is started by multiple, bilateral, painless swellings in the **upper part of the neck** 2 years ago by gradual onset & slowly progressive course.
- The condition was associated with **night sweat, night fever, loss of weight & loss of appetite**
- 2 days later multiple, bilateral swellings appeared in both **axilla**.
- 7 days later multiple, bilateral swelling appeared in both **groin**. The patient was admitted to Abbasia fever hospital for 7 days & investigated by urine, stool, CBC and chest X-ray & received medical treatment and fever disappeared.
- The patient is still complaining with dyspnea and cough, so admitted to chest hospital and received medical treatment. The symptoms disappeared but the swellings persist so admitted also to National cancer institute & received medical treatment in form of (4 types of drugs) so swelling decreased in size and persist until now.
- There are bilateral varicose vein & Rt. side hernia
- No symptoms suggesting pressure in **axilla** : in form of oedema, tingling numbness or claudication pain.
- No symptoms suggesting pressure in **groin** : same as axilla.
- No symptoms suggesting pressure inside **abdomen** : In form of renal pain jaundice or leg oedema.
- No symptoms suggesting **causes** as : Leukemia (bleeding tendency), 2ry syphilis (Skin rashes with genital ulcer) or IMN (skin rashes & glandular fever).
- ❖ **Past history :**
 - No past history about recurrence, No DM, No hypertension, No T.B, No Bilharziasis
 - There past history about Lt. side hernia operation
- ❖ **Family history :** No family history of similar condition (irrelevant)

❖ **Diagnosis :** Generalized lymphadenopathy most probably 2^{ry} T.B

Hodgkin's lymphoma

❖ **Personal history:** ...

❖ **Complaint :** Multiple bilateral swellings in the neck, axilla & groin 6 years ago

❖ **HPI:**

- The condition is started by multiple, bilateral, painless swellings in the **upper part of the neck** 6 years ago by gradual onset & slowly progressive course.
- At 1st appears in the neck then **axilla** & finally **groin** 1 week from the onset.
- The condition is associated by **fever 1 week & disappeared 10 days then returned in same manner**.
- The patient was admitted to Demrdash hospital & was investigated by C.B.C, CT chest, bone marrow aspiration from sternum & L.Ns biopsy from axilla, then received medical treatment in form of chemotherapy, so the patient's hair lost as complication.
- No symptoms suggesting pressure in **axilla**: In form of oedema, tingling, numbness or claudication pain.
- No symptoms suggesting pressure in **groin** : Same as axilla
- No symptoms suggesting pressure inside **abdomen** : In form of renal pain, jaundice or leg oedema.
- No symptoms suggesting pressure inside **abdomen** : In form of renal pain, jaundice or leg oedema.
- No symptoms suggesting **causes** as : Leukemia (bleeding tendency), 2ry syphilis (Skin rashes with genital ulcer) or IMN (skin rashes & glandular fever)
- ❖ **Past history :**
 - No past history about recurrence, No DM, No hypertension, No T.B, No Bilharziasis
- ❖ **Family history :** No family history of similar condition (irrelevant)

❖ **Diagnosis :** Generalized lymphadenopathy most probably Hodgkin's lymphoma

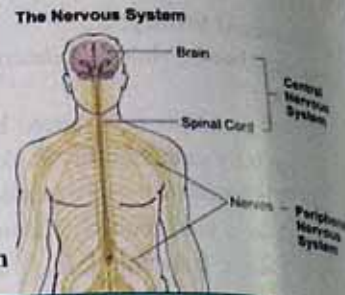
NEUROLOGY

❖ Classifications of nervous system:

A. Functional classification (Neurophysiology):

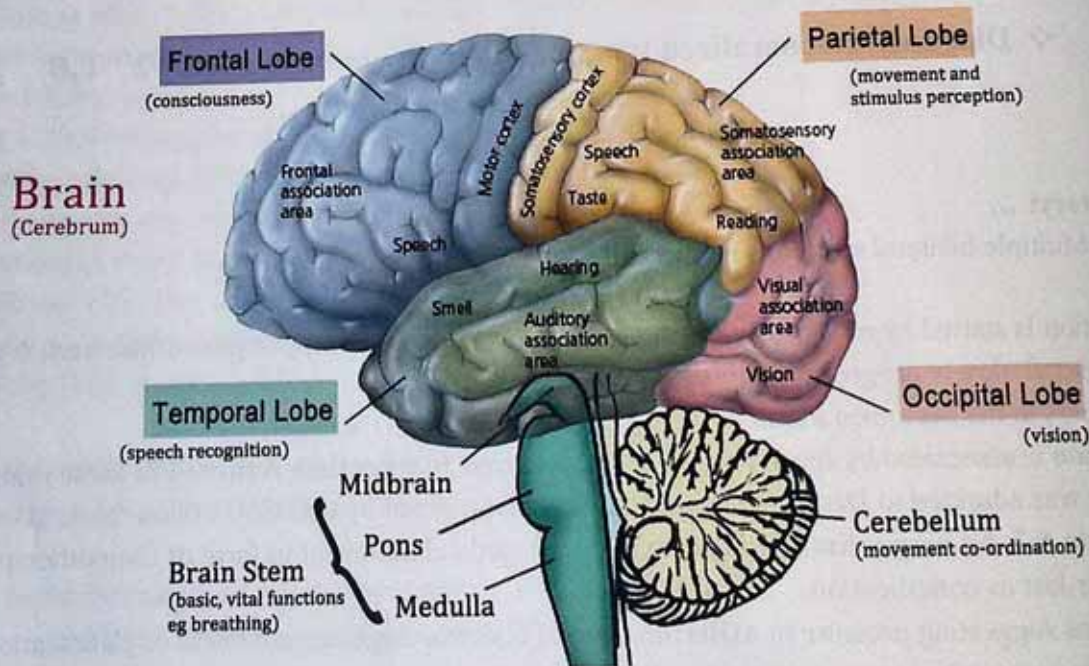
- Autonomic function : parasympathetic & sympathetic nervous system
- Somatic function : motor system & sensory system
- Psychological function.

B. Structural classification (Neuroanatomy): central & peripheral nervous system



1. Central nervous system (CNS)		2. Peripheral nervous system (PNS)	
A. Brain :	B. Spinal cord	A. Spinal nerves	B. Cranial nerves
⊗ Cerebrum	⊗ 31 segments	⊗ Number	12 pairs
⊗ Cerebellum		31 pairs	⊗ Origin
⊗ Brain stem :		Spinal cord	Brain & its stem
- Midbrain		⊗ Exit	
- Pons		Vertebral column	Skull (cranium)
- Medulla		⊗ Supply	
		• Cervical : UL	• Head & neck
		• Thoracic : trunk	
		• Lumbar: LL	
		• Sacral : LL	
		• Coxygeal : perineum	

Central nervous system (CNS)



A) Brain:

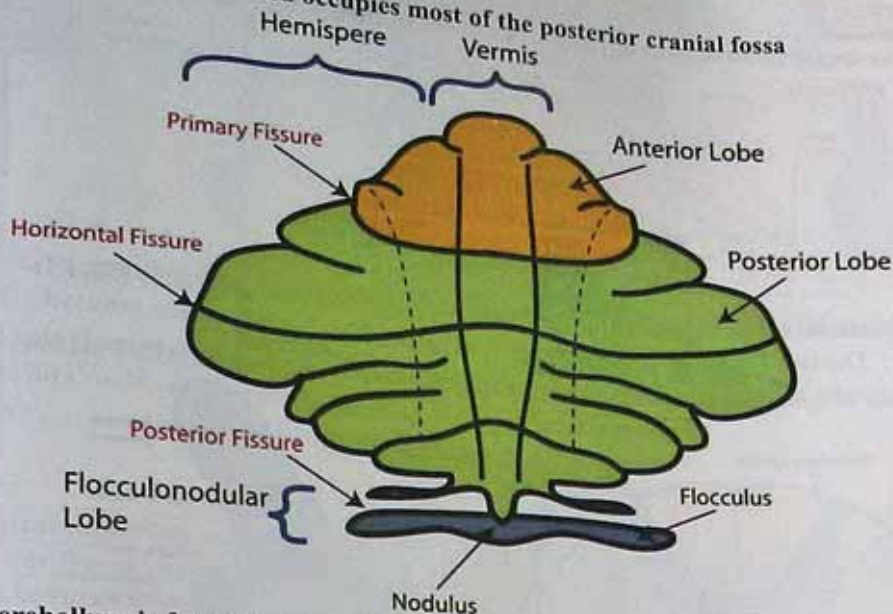
I. Cerebrum:

- ⊗ It is formed of 2 cerebral hemispheres, connected to each other by the corpus callosum, and to the upper part of the brain stem by the 2 cerebral peduncles.
- ⊗ The surface of each hemisphere is divided into 4 lobes:

1. Frontal	2. Parietal	3. Temporal	4. Occipital
------------	-------------	-------------	--------------

Cerebellum :

- ❖ **Site :** It lies behind the Brain Stem and occupies most of the posterior cranial fossa



- ❖ **Anatomy:** the cerebellum is formed of 2 main parts:

1. A midline central structure, known as the vermis.
2. Two lateral cerebellar hemispheres.

- ❖ **The cerebellum can also be divided in the following manner:**

- | | | |
|--------------------------|------------------|-------------------|
| 1. Flocculo-nodular lobe | 2. Anterior lobe | 3. Posterior lobe |
|--------------------------|------------------|-------------------|

- ❖ **Phylogeny :**

	A. Archicerebellum	B. Paleo-cerebellum	C. Neocerebellum
Anatomy	▪ Flocculo-nodular lobe	▪ Anterior lobe	▪ Posterior lobe
Development	▪ The earliest development	▪ The Next one	▪ The most recent
Function	▪ Maintenance of equilibrium	▪ Maintenance of muscle tone	▪ Coordination and regulation of the fine delicate voluntary motor activity
Connection to	▪ Vestibular apparatus	▪ Spinal cord	▪ Cerebral hemispheres
Connection through	▪ Vestibulo-cerebellar and cerebello-vestibular tracts	▪ Spino-cerebellar tracts.	▪ Dentato-rubro-thalamo-cortical tract.
Lesion	▪ Disturbance of equilibrium: - Trunkal ataxia on standing - Drunken gait on walking	▪ Disturbance of the muscle tone (hypotonia)	▪ Incoordination of movements.

III. Brain stem:

	Midbrain	Pons	Medulla
⊕ It is formed of:	CN 3, 4	CN 5, 6, 7	CN 9, 10, 11, 12
⊕ Motor nuclei of			



- ⊕ It is connected to the cerebral hemispheres by 2 cerebral peduncles and to the cerebellum, on each side, by the superior, middle & inferior cerebellar peduncles.
- ⊕ It contains groups of nerve cells (gray matter) intermingled with several ascending and descending fibers (white matter)

⊕ **N.B. :**

- CN: 1, 2 & 8 are sensory nerves having no motor nuclei, they concerned with special sensations, perceived in special areas of the cerebral cortex.

B) Spinal cord:

❖ **Site:** It lies in the spinal canal & ends at the lower border of the 1st lumbar vertebra.

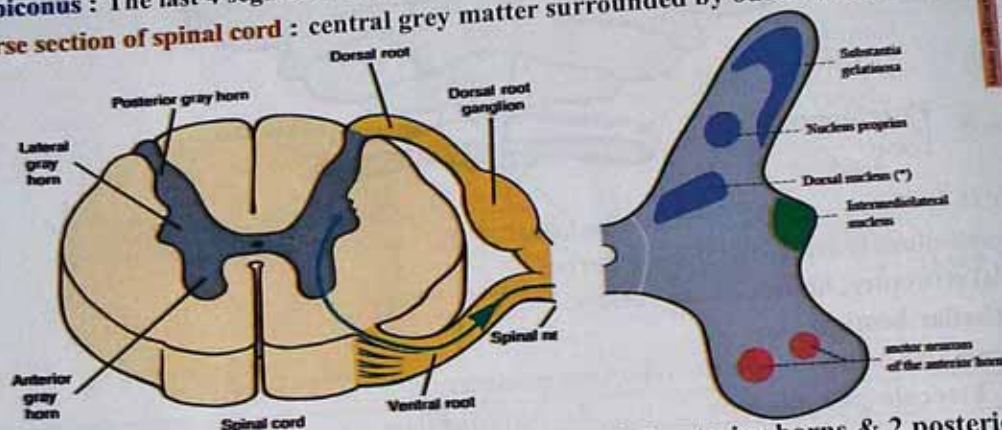
⊗ **Formed of:** 31 segments.

- 8 cervical
- 12 thoracic
- 5 lumbar
- 5 sacral
- 1 coxigeal

⊗ **N.B.:**

- **Conus medullaris:** The lowermost 3 segments of the spinal cord (S3, 4, 5) (S3,4,5)
- **Epiconus:** The last 4 segment above the conus (L4,5,S1,2)

❖ **Transverse section of spinal cord:** central grey matter surrounded by outer white matter:

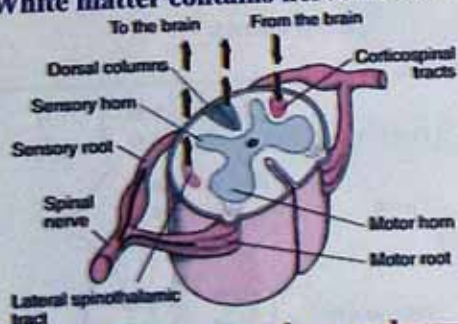


1. **Grey matter contains nerve cells:** arranged in H-shaped with 2 anterior horns & 2 posterior horns:

A. Anterior horn	B. Posterior horn
○ It's nerve cell bodies called anterior horn cells (AHC) ○ Has motor function	○ It's nerve cell bodies at the end called substantia gelatinosa of Rolandi (SGR) ○ Has sensory function
→ The fibers of grey matter extend outside the spinal cord forming the spinal nerves:	
○ Gives ventral (anterior) root of spinal nerve	○ Receives dorsal (posterior) root of spinal nerve

⚡ N.B.: lateral horn presents only in the thoracic & upper lumbar segments, has sympathetic function through sympathetic ganglion

2. **White matter contains nerve fibers** which forms ascending & descending nerve fibers arranged in tracts:



A. Ascending tracts:	B. Descending tracts:
○ Lateral spinothalamic ○ Ventral spinothalamic ○ Gracile & Cuneate ○ Spinocerebellar ○ Lissauer's tract	❖ Pyramidal: Corticospinal ❖ Extra-pyramidal: ○ Reticulospinal ○ Vestibulospinal, Tectospinal ○ Rubrospinal, Olivospinal

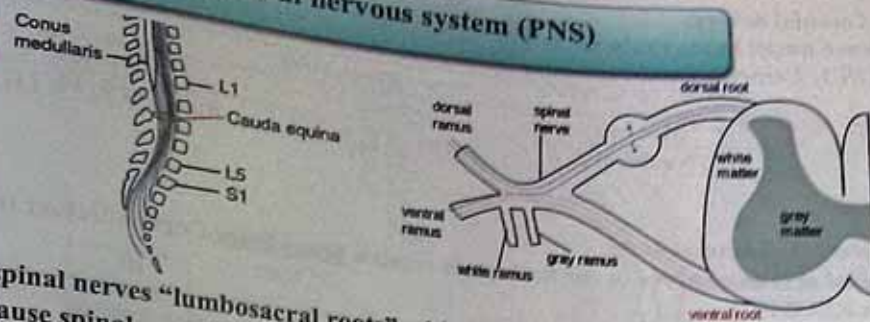
❖ **Relation between vertebrae and corresponding spinal cord segment:**

- Because bone growth is faster than soft tissue growth, vertebral column is longer than spinal cord which ends at L1 vertebra, So not every vertebra is opposite it's corresponding segment

❖ Spinal cord segment	❖ Vertebral column
○ From C1-C4	○ Subtract 1
○ From C5-T6	○ Subtract 2
○ From T7 - L2	○ Subtract 3
○ L3, L4	○ Opposite T12
○ L5 & sacral segments	○ Opposite L1
- E.g.	
○ Superior abdominal reflex = T10 Cord = T7 vertebra	
○ Ankle reflex = S1 cord = L1 vertebra	

A. Spinal nerves:

Number: 31 pairs



- Cauda equine: last 10 spinal nerves "lumbosacral roots" which fill the lower part of the spinal canal below the lower border of L1 because spinal cord is shorter than vertebral column
- Formation: each spinal nerve is formed by the combination of nerve fibers from the dorsal (posterior) and ventral (anterior) roots of the spinal cord.
- Type: mixed nerve
- Function :

A. Somatic function:

1. Ventral (anterior) root

- Composed of motor fibers "axons of AHCs"
- Its cell bodies lie in specific areas of the spinal cord itself.

2. Dorsal (posterior) root

- Composed of sensory fibers
- Its cell bodies lie in a spinal ganglion (dorsal = posterior root ganglion) that is outside the spinal cord

B. Autonomic function :

- Sympathetic fibers through thoracolumbar spinal nerves (T1-L2 levels)
- Parasympathetic fibers in sacral plexus (S2, S3 and S4 levels)

B. Cranial nerves = CN

Number: 12 pairs

1. Olfactory	2. Optic
3. Oculomotor	4. Trochlear
5. Trigeminal	6. Abducent
7. Facial	8. Vestibule-cochlear
9. Glossopharyngeal	10. Vagus
11. Accessory; has 2 part:	12. Hypoglossal
- Cranial part	
- Spinal part	

→ N.B.: Groups :

▪ Ocular group	▪ Bulbar group
CN 3, 4, 6	CN 9, 10, 11, 12

Function: Nerve supply to head & neck

A. Somatic Function :

1. Pure sensory nerves: special senses	2. Pure motor nerves	3. Mixed nerves
• CN 1 : smell • CN 2 : vision • CN 8 : hearing	• CN: 11, 12, Ocular group (3, 4, 6)	• The others

B. Autonomic function:

- Sympathetic supply through sympathetic chain
- Parasympathetic supply through parasympathetic ganglions

→ N.B.: Cranial nerves act on 2 types of muscles in head & neck

1. Pulling muscles	2. Pushing muscles
• Mouth : Retractor anguli • Uvula : Muscles of uvula	• Tongue لسان • Mandible فك
If both of them are intact : target in the middle If one of them is weak : deviation :	
• Deviation to the normal (healthy side)	• Deviation to the diseased side فكسان عيان

❖ **Nuclei of cranial nerves:**

- All CN have nuclei in the brain stem
- Except CN 1, 2 emerge from cerebrum so they are not considered CN but they are considered as tracts

❖ Brain stem		
▪ Mid brain :	▪ Pons	▪ Medulla
○ Nuclei of 3, 4	○ nuclei of 5, 6, 7, 8	○ nuclei of 9, 10, 11, 12

❖ **Cortical control of cranial nerve :**

- Motor nuclei of cranial nerve in the brainstem receive fibers from Cortico-bulbar tract of pyramidal tract of both sides EXCEPT :

- Lower part of CN 7
- CN 12 (unilateral pyramidal supply in 50 % of people)
→ Which have unilateral pyramidal fibers from the opposite side

❖ **Lesion of CN are either :**

- Upper Motor Neuron Lesion (UMNL) : pyramidal lesion
- Lower Motor Neuron Lesion (LMNL) : nucleus , nerve, muscle

❖ **Golden rule :**

→ Unilateral paralysis in a CN with bilateral pyramidal tract supply is LMNL

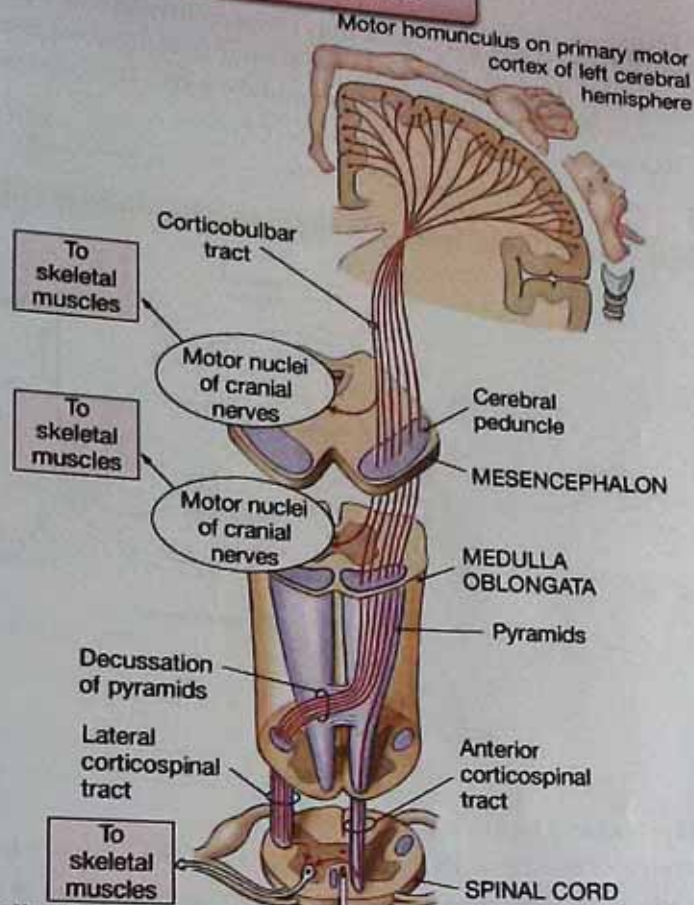
Motor system; consists of 4 main components:

1. Pyramidal (Δ) System (U.M.N.)	2. Extrapyrarnidal (extra Δ) System	3. Cerebellar System	4. Lower Motor Neuron (L.M.N.)
❖ Origin			
▪ It originates in the motor area (4) & premotor area (6) ▪ Terminates at the anterior horn cells (A.H.C.) of the different levels of the spinal cord	▪ It originates from centres situated at various levels of C.N.S. mainly the Basal Ganglia	▪ It is composed of the Neo, Archi & Paleo-cerebellum	▪ It is formed of A.H.C.s & peripheral motor nerves
❖ Control:			
▪ Opposite half of the body.	▪ Opposite half of the body.	▪ Same half of the body.	▪ Same half of the body.
❖ Function:			
▪ Initiation of voluntary motor activity ▪ Inhibition of muscle tone ▪ Inhibition of deep reflex	▪ Regulation of voluntary motor activity ▪ Regulation of muscle tone ▪ Regulation of emotional & associative movement	▪ Coordination of voluntary motor activity ▪ Regulation of muscle tone ▪ Regulation of equilibrium	▪ Transmit the motor impulses to the voluntary muscles

Upper & lower motor neuron systems = Pathway of voluntary motor impulse

- For a voluntary muscle to move, a nerve impulse originates from the opposite motor area to pass into main neurons UMN (Δ tract) & LMN

1. Upper Motor Neurone (U.M.N.) = pyramidal tract



❖ **Origin:** Betz (pyramidal) cells of the motor area (area 4) & to a lesser extent in the cells of the premotor area (area 6).

❖ **Pathway :**

1. Axons of these cells descend in the depth of the cerebral hemisphere in the **corona radiata**
 2. Pass in the **internal capsule** (genu & ant. 2/3 of posterior limb)
 3. Continue their descent in the midbrain, pons & medulla in the **brain stem**, some of pyramidal tract leave it to supply **motor nuclei of the cranial nerves** of both sides except the lower half of the facial nucleus & the hypoglossal nucleus (in 50% of people) which are supplied only from the opposite Δ tract.
- These fibers are known as **corticobulbar** as they do not reach the spinal cord.
4. In the **lower medulla**, 80% of the fibers **decussate** to descend in the white matter of the opposite side of the spinal cord, while the remaining fibers (20%) descend directly in the white matter of the same side.
- This pathway starting from the cells of the cortex down to the spinal cord is known as the **pyramidal Tract**.
- The fibers of the pyramidal tract terminate at different levels of the **A.H.C.s of the spinal cord** is known as **Corticospinal tract**

❖ **N.B.:**

- Body is represented in motor area 4 (precentral gyrus) **upside down**, with the face at lower part & foot at the top
- Skilled & complex movements e.g. Those involving speech, face & hands are specially widely presented

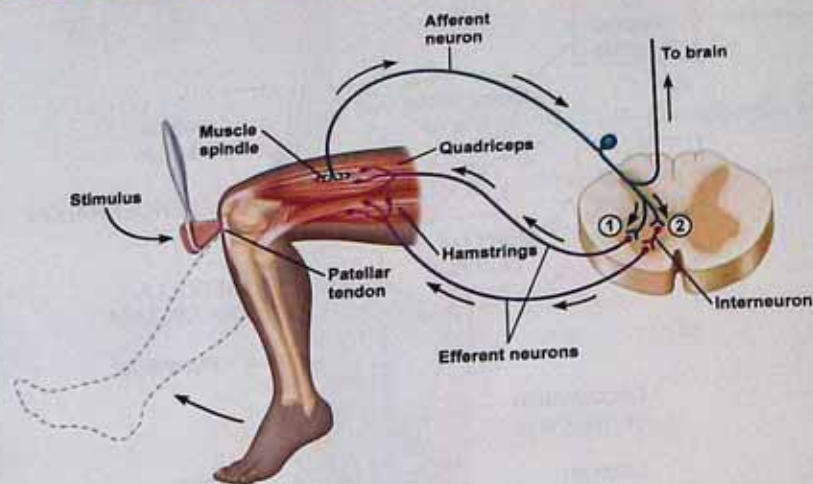
2. Lower Motor Neurone (L.M.N.)

1. **A.H.C.s:**
 - They receive the voluntary motor impulse from the Δ tract.
2. **Peripheral Motor Nerve** carrying the motor impulse from A.H.C. to the voluntary muscle through motor end plate

❖ N.B.:

- Spinal cord AHCs receive pyramidal fibers from contralateral side only
- The motor nuclei of the cranial nerves are similar in function to the A.H.C., as they form the cell bodies of the L.M.N. of the cranial nerves. Thus, lesion of a cranial nerve nucleus, like lesion of an A.H.C. is a L.M.N. lesion.

👉 To know the UMN lesion & LMN lesion, we should understand the control of muscle tone & stretch reflex first.



❖ **Muscle tone :**

- This is a spontaneous (involuntary) local axon stretch reflex.
- Due to constant slight stretch of muscle as its length shorter than the distance between its origin & insertion.
- This stretch stimulates some muscle spindles which send excitatory impulses through the afferent sensory nerve & the dorsal root to the A.H.C.
- The A.H.C.s send motor impulses through the anterior root & the efferent motor nerve to the muscle.
- This results in continuous reflex subtetanic contraction of the muscle = Muscle Tone
- Function of muscle tone: which is important for the nourishment of the muscles & the posture of the body.

❖ **Tendon Jerk (Deep Reflex):**

- This is an induced local axon stretch reflex.
- It is induced by tapping the tendon of the muscle with a hammer. This tap stretches the muscle with synchronous stimulation of all muscle spindles & activation of the local axon reflex (as in Muscle Tone), resulting in a brief contraction of the muscle.

👉 **Control of muscle tone & stretch reflex :**

- Both are inhibited by higher control, through the pyramidal & extrapyramidal systems.

👉 **Lesion:**

- U.M.N.L (Δ Lesion) results in loss of inhibition of the intact reflex arc leading to increased muscle tone (hypertonia; spasticity or rigidity) & exaggeration of deep reflexes (hyperreflexia) below the level of the lesion with no wasting of the muscle.
- L.M.N.L. results in interruption of the reflex arc leading to decreased muscle tone (hypotonia; flaccidity) & diminution of deep reflexes (hyporeflexia) at the level of the lesion, with wasting of the muscles.

❖ **Clonus:**

- Occurs in severe Δ lesion (stretch reflex arc is no longer inhibited)
- **Organic Clonus:** Induced by sudden sustained stretch of muscle tendon, resulting in a series of rhythmic contractions. It stops with relief of tendon stretch.
- Elicited in ankle, patella & wrist.
- Should be differentiated from hysterical clonus: where it continues after relief of tendon stretch

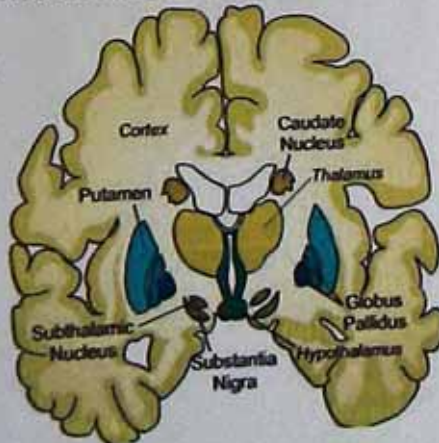
❖ **UMNL & LMNL:**

	UMNL	LMNL
	❖ Pyramidal tract lesion	❖ Lesion
		❖ Weakness
Effect	- Paralysis or weakness - Loss of skilled voluntary movement	Lesion of AHC or peripheral nerve or posterior root or muscle.
Site	Below the level of the lesion	At the level of the lesion
Distribution	- Distal > Proximal - Progravity > antigravity - Abductors > adductors	All muscles supplied
	❖ Tone	
Effect	Hypertonia "release phenomenon" (spasticity) "clasp knife"	Hypotonia (flaccidity)
Site	Below the level of the lesion	At the level of the lesion
Distribution	- Adductors > abductors - Antigravity > Progravity	All muscles supplied
	❖ Reflexes	
Superficial	- UMNL above level of segmental supply of reflex	- LMNL affecting the reflex arc itself
Deep	Hyperreflexia below the level of the lesion	Hyporeflexia at the level of the lesion
Pathological	Present	Absent
Clonus	may be present	Absent
Plantar reflex	Extensor response = Babinski sign	Flexor response = Normal response
Atrophy	❖ No or disuse atrophy : - Late - Slight - Diffuse	❖ Yes: - Early - Marked - Localized
Trophic changes	Never	±
Fasciculations	Never	± (affection of AHCs i.e. irritative lesion)

Extra-pyramidal system

❖ **Definition:** It includes all fibers that can influence the motor end plate activity and do not pass in the pyramidal tract.

❖ **This system is formed of the following areas:**



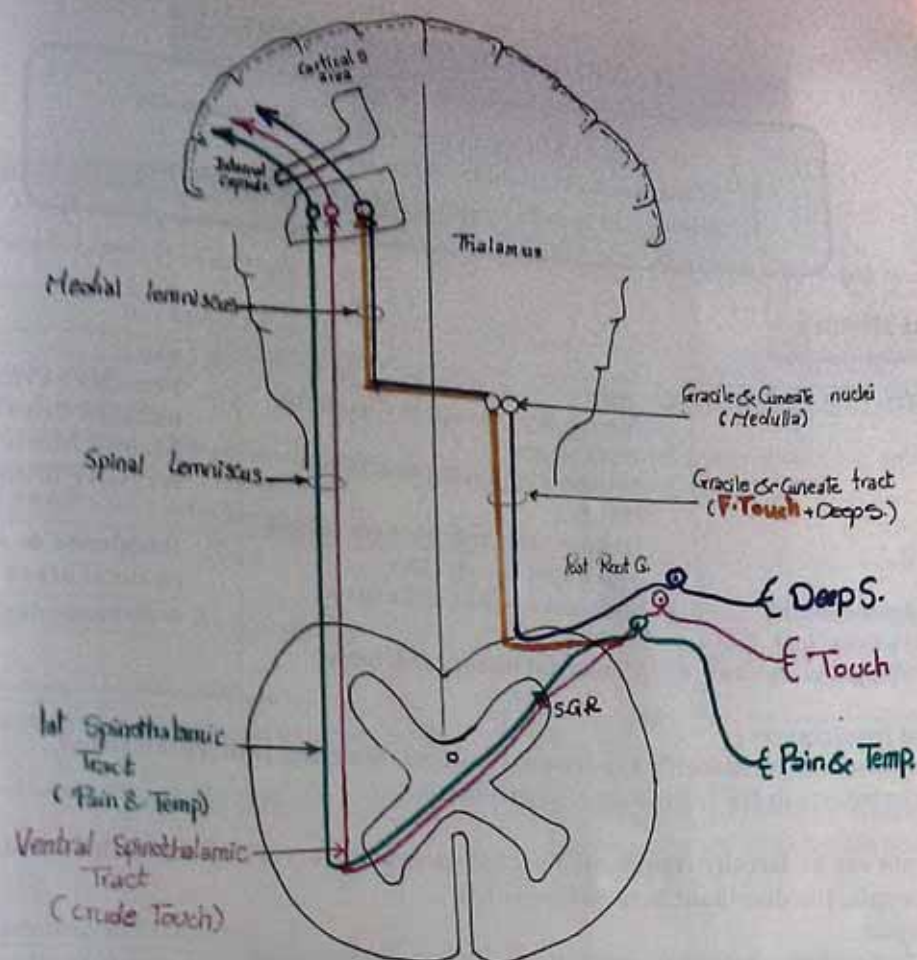
1. **Cortical level:** all lobes share but mainly in the frontal lobe.
2. **Telencephalic level (Basal ganglia):** including the caudate nucleus and the lentiform nucleus (which is formed of the globus pallidus and the putamen).
3. **Diencephalic level:** thalamus, hypothalamus and subthalamus.
4. **Mesencephalic level (Midbrain):** red nucleus and substantia nigra.
5. **Pontine level:** pontine reticular nuclei.
6. **Cerebellar level.**

❖ Functions of the extrapyramidal system	❖ Lesion of extrapyramidal system:
• Regulation and integration of voluntary motor activity.	• Hyperkinesia i.e. static tremors which may be of two main types: ○ Rhythmic and regular as in Parkinsonism. ○ Dysrhythmic and irregular as in chorea, athetosis and dystonia. → N.B.: These tremors increase with emotional stress, anxiety and fatigue and disappear during sleep and during active voluntary movements.
• Regulation and maintenance of the muscle tone.	• Hypertonia = rigidity in parkinsonism • Hypotonia in chorea • Alteration of hyper & hypotonia in athetoses & dystonia
• Regulation and maintenance of emotional and associative movements.	• Reduction : mask face in parkinsonism • Exaggerated (excess grimacing) in chorea

Sensory system

❖ Sensations, in general are classified into:

1. **Somatic sensations:** are conducted to the CNS via somatic nerves; they include:
 - A. **Superficial sensations:** Pain, Temperature, Touch.
 - B. **Deep sensations (Proprioceptive):**
 - Vibration sense, Joint sense, Muscle sense, Nerve sense.
 - C. **Cortical sensations:**
 - Tactile localization, 2 points discrimination, Stereognosis, Graphosthesia, Perceptual rivalry
 2. **Visceral sensations:** all sensations from internal viscera reaching the CNS via the autonomic nerves.
 3. **Special sensations:** including vision, hearing, smell & taste reaching the CNS via the cranial nerves.
- ❖ **Pathway of superficial & deep sensations:**



	Superficial sensations			A. Deep sensations
Type of sensation:	Pain, Temperature	Crude "simple" touch	Fine touch	• Vibration sense, Joint sense, Muscle sense, Nerve sense.
Receptors in	Skin			Deep structures
1 st order neuron	▪ The cell of the Posterior root ganglion & it's axon. ▪ This axon is divided into : - The lateral branch forms the afferent sensory never . - The medial branch enters the spinal cord to ascend a few segment forming ... ⊕ Lissauer's tract ▪ Gracile & Cuneate tracts within the posterior column of the same side (along with fibers carrying fine touch) ↳ Gracile Tract: carries fibers from lower ½ of body and lies medially. ↳ Cuneate Tract: carries fibers from upper ½ of body and lies laterally			
Site of relay of 1 st order neuron	▪ SGR			▪ Gracile & Cuneate nuclei in medulla
2 nd order neuron	▪ Cells of Substantia Gelatinosa of Rolandi (S.G.R.) in the post horn of the spinal cord			▪ Gracile & Cuneate nuclei in medulla
Crossing to opposite side in :	Spinal cord			Medulla
& ascend in ... Tract	Lateral spinothalamic	Ventral spinothalamic		Gracile & Cuneate
Then in ... Lemniscus in brain stem.	Lateral			Medial
3 rd order neuron:	▪ Thalamus of the opposite side area 1, 2, 3 in parietal lobe ▪ Cortical sensory area of the opposite side			
End in:				

NEUROLOGICAL HISTORY

❖ Aim of history in neurology → full diagnosis

1. Main diagnosis (lesion) : from HPI
2. Etiology : From Personal , HPI, Past & Family History

A. Personal History

1. Name: الاسم ثلاثي	2. Age: ○ Young age: myopathy, embolic hemiplegia ○ Middle age: multiple sclerosis (MS) ○ Old age: thrombotic hemiplegia	3. Sex : ○ Females : Friedreich's ataxia, collagen diseases ○ Males : Marie's ataxia , X linked recessive disorders
4. Occupation: ○ Printers for lead neuropathy ○ Drivers & porters : cervical spondylosis, paraplegia & cauda equina	5. Marriage: for sterility, impotence, still-births (as in syphilis) 6. Menstrual history if female.	7. Residence & address: pellagra in rural areas (deficient in maize)
8. Habits of medical importance: Alcoholic (Peripheral neuritis, Karsakoff's syndrome, tremors), Smoking (atherosclerosis), Contraceptive pills contribute to DVT, headache, depression		
9. Handedness: • The centers in brain are bilaterally represented except speech centers which lie in the dominant hemisphere • In right handed people, the dominant hemisphere is left • In left handed people: ○ Mostly 70 % dominant hemisphere is left ○ Few (15%) dominant hemisphere is right ○ Few (15%) bilaterally represented		

B. Complaint + duration: ؟ إي اللي جابك المستشفى

❖ Common complaints :

▪ Heaviness on (one side e.g. right) upper & lower limbs	▪ Heaviness of both lower limbs	▪ Heaviness of both upper & lower limbs	▪ Shaking movement of both upper & lower limbs
▪ Loss of sensation in both hands & feet	▪ Staggering gait	▪ Inability to reach mouth with spoon properly during eating	▪ Inability to stand up from sitting position

C. History of present illness = HPI ؟ المرض بدأ معاك إزاي , آخر مرة كنت كويس فيها إمتى ؟

2 سيستم : حركة وإحساس	1. Symptoms of Motor affection	2. Symptoms of Sensory affection
2 دماغ : أعصاب وضغط	3. Symptoms of Cranial nerve affection	4. Symptoms of increased Intracranial tension
2 يخرجهم : كلام وحمام	5. Symptoms of Speech affection	6. Symptoms of Sphincteric & sexual troubles
2 آخرين : تحاليل وأجهزة	7. Investigations & treatment received	8. Symptoms of other systems

N.B.:

تكتب في ال complaint بكلام العيان
تكتب في H/O of present illness عادي

Analysis of any complaint : OCD & associated symptoms

تحليل ال OCD & associated symptoms مهمين جدا في النورول لانهم يساعدوني اني اعرف ال etiology فهنتكم عنهم بالتفصيل شوية :
1. Onset : من اول عرض حتى اكتمال الصورة النهائية للمرض :

A. Acute onset :	B. Gradual onset:
a) Dramatic onset: ثواني (من ثواني لايام 14 يوم) - trauma & embolism b) Apoplectic onset: دقائق hemorrhage (apoplexy) c) Sudden onset : ساعات : thrombotic d) Rapid onset : ايام : inflammation	▪ أكثر من 14 يوم (اسبوع - شهور - سنين) ▪ Tumors ▪ Space occupying lesion e.g. tuberculoma ▪ Degeneration

ترتيب أسباب ال hemiplegia طبقا لل onset ← EAT IT : E: Embolism ▪ A: Apoplexy ▪ T:Thrombosis ▪ I: Inflammation ▪ T: Tumor

2. Course : من اكتمال الصورة النهائية حتى الآن :

Progressive: more severe symptoms or new symptoms	Regressive	Stationary
Remittent: in between attack the patient is ill e.g. DS	Intermittent: in between attack the patient is free e.g. Transient ischemic attack (TIA)	

3. Associated symptoms: Relation to:

Inflammation: Fever	Traumatic: Trauma	Hemorrhage: Vomiting ,convulsions, coma
---------------------	-------------------	---

✗ If negative: not associated with either fever, trauma, vomiting, coma, or convulsion

✗ N.B.: diagnosis of disseminated sclerosis (DS) is by 2 things:

1. Remittent course
2. Multi-focal (white matter only in CNS)

1. Symptoms of motor affection (weakness, involuntary movements, incoordination)

1) Weakness شلل - تقل - ضعف

➤ Heaviness or weakness

- Analysis : OCD : for etiology (see before), Character & distribution for the lesion

Character:

Degree : paresis (weakness) or complete paralysis

شلل كلي ولا ضعف بس ؟

Tone :

Hypertonia عضلاتك ماسكة - مخشبة - ناشفة - شدة

➤ Stiff

Hypotonia ولا سايبة - مرخية

➤ Flaccid or flail

Atrophy: عضلاتك خست ولا زي ما هي قبل العيا ؟

➤ Muscles are wasted

لو قال خست: بعد قد اي من العيا ؟ خست جامد ولا قليل ؟

Fasciculations عضلاتك بترف ولا لا ؟

➤ Muscle Twitches

Trophic changes:

شعرك بيقلع ولا لا ؟ ضوافرك بتقصف ولا لا ؟

➤ Falling of hair & Brittling of nails

If negative, don't mention it.

Distribution: all questions are asked indirectly but the answer is written directly :

UL or LL or both

الضعف في ايديك ولا رجلك ولا ايدك ورجلك ؟

لو قال ايدي ورجلي : بدأ في مين الأول ؟ دلوقتي مين أضعف ؟

Unilateral or bilateral

يمينك ولا شمالك ؟ ولا يمينك وشمالك ؟

لو قال يميني وشمالي : بدأ في مين الأول ؟ دلوقتي مين أضعف ؟

Distal or proximal:

في ال UL : أسهلك تعصر ليمونة ولا تسرح شعرك ؟

في ال LL : أسهلك تلبس شيشب ولا تطلع السلم ؟

	Distal	Proximal
UL	تعصر لمونة - بتعرف تكتب - تشتغل بالإبرة - تسيح	تسرح شعرك - يسوق - يشيل حاجة - يليف نفسه
LL	تلبس الشيشب - تنزل السلم	تطلع السلم

Extensors or flexors :

في ال UL : أسهلك تقفل الدرج ولا تفتحه ؟

في ال LL : أسهلك تلبس البنطلون ولا تفرد رجلك فيه ؟

	Extensors	Flexor
UL	تقفل الدرج	تفتح الدرج
LL	يفرد رجليه	تلبس البنطلون

Abductors or Adductors :

في ال UL : أسهلك تلبس القميص ولا تشيل حاجة تحت باطك ؟

في ال LL : تشيل رجل من على رجل ولا تحط رجل على رجل ؟

	Abductors	Adductors
UL	تلبس قميص	تشيل حاجة تحت باطك
LL	تشيل رجل من على رجل	تحط رجل على رجل

2) Involuntary movements إيدي بترعش

➤ Shaking movement

Character :

Static مع الثبات In parkinsonism, chorea

Regular, Rhythmic منتظمة In parkinsonism

Hypertonia مخشبة In parkinsonism

What increases? E.g. stress

What decreases? E.g. sleep

Or kinetic مع الحركة In cerebellar lesion

Or irregular ملخبطة dysrhythmic In chorea

Hypotonia سايبة In chorea

Distribution: all questions are asked indirectly but the answer is written directly :

UL or LL or both

إيديك ولا رجلك ولا إيديك ورجلك ؟

Unilateral or bilateral

يمينك ولا شمالك ولا يمينك وشمالك ؟

Distal or proximal:

الرعدة أكثر ناحية كفك ولا كتفك ؟ مشط رجلك ولا فخذك ؟

Involuntary movement in head & neck

في رأسك ورقبتك ولا لا ؟

In other parts of the body

أي حدة تأتي في الجسم ؟

3) Incoordination = ataxia

- ASKED ONLY IF THERE IS NO WEAKNESS :

1. Cerebellar ataxia (Long case):

A. LL: early: يمشي يطوح

➤ Staggering gait

B. UL: late

لما آجي أكل أو أشرب الملعقة ترعش كل لما تقرب

➤ Inability to reach the mouth with spoon properly during eating

2. Sensory ataxia (part from P.N. case):

A. LL: لما آجي أغسل وشي أقع

➤ Unsteadiness during eye closure

B. UL:

في السيدات : لا يمكن ربط السوستة الخلفية للفساتان

في الرجال : لا يمكن ربط الكرافة بدون مرآة

2. Symptoms of sensory affection (superficial, deep & cortical sensation)

1) Superficial sensation:

❖ Analysis: OCD: for etiology (see before), Character & distribution for the lesion.

Character

A. Irritation (early stage) في وجع أو شكشكة أو تميل ؟

1. Pain وجع (for analysis see cardiology):

➤ Pain

i. Diabetic Peripheral neuropathy:

o Burning, nocturnal pain

ii. Root (radicular pain): الوجع محزمني, لافني

2) Deep sensation :

وانت ماشي بتحس انك ماشي على حاجة صلبة ولا على قطن ؟

➤ Negative : صلبة

➤ No symptoms suggestive of deep sensory affection.

➤ Positive : قطن : for sensory ataxia (Positive Rombergism) :

➤ The patient feels as if he walks on cotton

- Site: along it's dermatome
 Character : stitching
 Increased by: coughing, sneezing, straining
 Decreased by: rest & support
 Causes :
 Paraplegia :
 - girdle pain
 Value in leveling & etiology (extramedullary)
 Cauda equine (lumbo-sacral root)
 Cervical spondylosis
 Paresthesia عندك شحنة أو تميميل ; abnormal sensation : tingling & numbness
 Destructive lesion (late stage) : الاحساس قل ولا طبيعي ؟
 Hypoesthesia قل الاحساس
 Anesthesia انعدم نهائيا
 Distribution: UL or LL or both, unilateral or bilateral, proximal or distal

3) Cortical sensation

ASKED ONLY IF BOTH SUPERFICIAL AND DEEP SENSATION ARE INTACT :

- لما تحط حاجة في جيبك تعرف اللي فيه ولا لازم تطلعها وتبص عليها ؟
 If negative: ببعراف الحاجة
- No symptoms suggestive of cortical sensory affection
- If positive : لازم بطلع الحاجة ويبص عليها
- Patient cannot recognize things in his pocket unless he sees it.

3. Symptoms of Cranial nerve affection

Cranial nerve affection			
CN 1: - حاسة الشم عندك قلت ولا طبيعي؟ - Anosmia , Parosmia	CN 2: a) Acuity of vision: b) Colored vision: - النظر ضعف ولا طبيعي؟ - أي عين؟ - بتعرف تميز الألوان من بعضها ولا لا؟ - وأنت ماشي بتخط في الناس والعواميد ولا لا؟	CN 3, 4, 6: ocular nerves: a) Ptosis - جفك سقط ولا طبيعي؟ لو آه - انهي ناحية؟ b) Squint - عينك احولت؟ لو آه انهي ناحية - ولبره ولا جوه؟ c) Diplopia - بتشوف الحاجة اتنين ولا طبيعي؟	CN 5: trigeminal a) Motor : difficult mastication - في صعوبة في المضغ ولا طبيعي؟ b) Sensory : face sensation - Irritation : - في وجع او شحنة او تميميل في وشك ولا طبيعي؟ - Destruction : - الاحساس في وشك قل ولا طبيعي؟
CN 7, facial : a) Upper motor : - Orbicularis oculi : - لما بتفصل وشك وترر على عينك , في صابون بيدخل؟ لو آه انهي ناحية؟ - عينك بتدمع زيادة ولا طبيعي؟ - لو آه انهي ناحية؟ b) Lower motor: - Buccinator : - لما بتاكل , الأكل بيتجمع في بقتك ولا طبيعي , لو آه أي ناحية؟ - Orbicularis oris - ريقك بينزل من بقتك ولا طبيعي , لو آه أي ناحية؟ - Retractor angulator - بقتك اتعوج على ناحية ولا طبيعي؟ لو آه أي ناحية؟ c) Sensory : - Taste in anterior 2/3 of the tongue - بتعرف تذوق الأكل بطرف لسانك ولا لا؟		CN 8: Vestibulocochlear a) Cochlear part : - Irritating : tinnitus - فيه وش في الودان ولا طبيعي - Destruction: diminished hearing, deafness - السمع تقل ولا طبيعي؟ b) Vestibular part: - بتحسن ان الأرض بتلف بيك ولا لا؟ (متقولش انته دايع ولا لا)	
CN 9, 10, { 11; cranial accessory}:			
a) Palate : - Nasal tone صوتك اخنف ولا لا - Nasal regurge - لما بتشرب فيه , بترد من مناخيرك ولا لا	b) Pharynx - Dysphagia - في صعوبة في البلع ولا لا - Chocking attacks - بتشرق كثير ولا لا	c) Larynx - Dysphonia (hoarseness of voice) - صوتك اتبج ولا لا - Dysarthria - في صعوبة في النطق ولا لا	
CN 11: spinal accessory - Sternomastoid بتعرف تحود رقبتك شمال ويمين ولا لا - Trapezius كتفك سقط ولا طبيعي؟ أي ناحية؟			CN 12: hypoglossal - لسانك اتعوج على ناحية ولا لا , أي ناحية؟ - في صعوبة في النطق ولا لا؟
هل عندك صداع مستمر لا يستجيب للمسكنات؟ هل حدث لك قهر؟ هل عندك غثاقل؟			

4. Symptoms of increased intracranial tension	• Projectile vomiting	• Blurring of vision	• Convulsions & coma
Persistent headache Severe bursting At the early morning (not relived by sleep) Partially relived by the vomiting	At the peak of headache Not related to meals, not preceded by nausea		

5. Symptoms of Speech affection: في مشاكل في الكلام ولا طبيعي ؟

6. Symptoms of Sphincteric troubles

- Defecation
- Micturition :
 يتبول على نفسي - مش قادر أتحمك في البول
 Incontinence
 Retention with overflow or automatic bladder or

Symptoms of Sexual troubles:

- Impotence in DM , multiple sclerosis
- Degree of impotence :
 Partial : can give birth
 Complete : can't give birth

nocturnal enuresis > Hesitancy: البول لازم اتحاييل عليه شويه - البول بيتأخر عندي > Difficulty in initiation of micturition وأنا رايح الحمام البول يسبب مني - البول بيسبقتني > Difficulty in holding urine when he feels the desire	Analysis : عندك مشاكل في الانتصاب أثناء المعاشرة الزوجية ولا ؟ لو في - مرات ومرات ولا على طول ؟ لو اه - الانتصاب الصباحي ضعف ولا طبيعي ؟ لو اه - بتأخذ أدوية ولا ؟
7. Investigation & treatment received عملت فحوصات اي ؟ واخذت علاج اي ؟ • CT brain, MRI, carotid duplex • ECG, echocardiography • Blood sugar, lipid profile • Physiotherapy, vitamins, antiplatelet drugs, anticoagulant drugs.	8. Symptoms of other systems هل عندك مشاكل تانية في جسمك ؟ وتأخذ كل سيستم وتسال العيان على الأعراض الكبيرة اللي فيه بسرعة اوي

D. Past history as usual but focus on:

Diseases 1. Rheumatic fever : embolic hemiplegia, SA hemorrhage (in SBE), chorea 2. Chronic suppuration : bronchiectasis & otitis media in brain abscess, facial palsy 3. Diabetes: Peripheral neuritis 4. Hypertension: in stroke 5. TB: TB toxemia in paraplegia (Pott's), cerebellar ataxia (tuberculoma) 6. Syphilis: chancre, recurrent abortions in sensory ataxia (Tabes dorsalis)				
Operations & drugs				
o Convulsion : ambilhar o 8 th CN: streptomycin	o Peripheral neuritis : INH, streptomycin	o Parkinsonism : major tranquilizer o 2 nd CN: ethambutol	o Myopathy : vincristine, steroids	o Cerebellar ataxia : hydantoin
Similar attacks : in DS & T.I.As Trauma: disc prolapse, paraplegia, cauda equina lesion Fever: meningitis, encephalitis, transverse myelitis				

E. Family history as usual & asked for congenital & heridofamilial diseases

DISCUSSION ON HISTORY

What are the causes of micturition disturbance in neurological case?

→ Causes of neurogenic bladder (Lesion must be bilateral):

- o UMNL : Paraplegia (nerve hemiplegia)
- o LMNL:

Segment : Conus lesion	Root: Cauda equina lesion	PN: Peripheral neuropathy, DM
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How can TB cause paraplegia ?

Mechanical :	Inflammatory :	Vascular :
▪ Bone : Pott's disease ▪ Soft tissue : psoas abscess	▪ Transverse myelitis ▪ TB pachymeningitis	▪ Endarteritis obliterans of the anterior spinal artery

What is the effect of Bilharziasis in neurological patient ?

- Bilharsoma : rare
 - o Space occupying lesion, affecting spinal cord causing paraplegia
- Ambilhar (niridazole): Antibilharzial drug causing convulsions

What is the difference between congenital disease & heredofamilial disease ?

Congenital disease	Heredofamilial disease
▪ The patient is born with anatomical or physiological defect even if there are no symptoms e.g. Congenital heart disease ▪ Not necessarily positive family history	▪ The patient is born anatomically & physiologically normal ▪ The symptoms appear at certain age e.g. Cerebellar ataxia ▪ Positive family history

❖ What is the incidence of impotence?

Incidence	Psychic	Organic
In the past	90 %	10 %
In the present time	50 %	50 %

❖ What are the causes of impotence?

1. Psychic : anxiety neurosis
2. Organic :

A. Nervous

→ Erection is a reflex having :

▪ Afferent : posterior root ▪ Center : S2,3,4 ▪ Efferent: anterior root

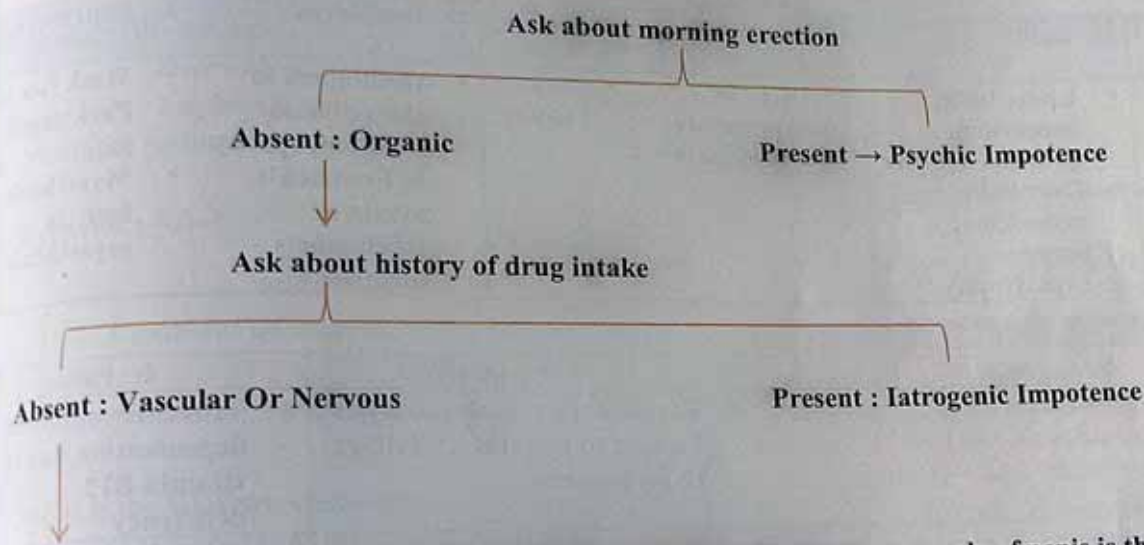
↳ So any lesion in :

▪ Segment : Conus lesion ▪ Root : Cauda equina lesion ▪ PN: Peripheral neuropathy, DM

B. Vascular "ischemia of penis": Le Rich syndrome, Sympathectomy of L1

C. Iatrogenic e.g. H2 blocker for treatment of Peptic ulcer & antihypertensive drugs except ACEI

❖ How to differentiate between causes of impotence?



Testicular sensation test "pressure on testis": it depends on that nerve supply of penis is the same of testis:

Intact sensation in vascular causes Impaired sensation in nervous causes

❖ What is the degree of impotence?

- Complete : no erection of any degree at any time, can't give birth
- Partial: erection or semierrection occur, can give birth

❖ What are the causes of impotence in diabetic patient?

○ Diabetic peripheral neuropathy "nervous"

○ Diabetic ischemia "vascular"

○ Usually diabetes is associated with atherosclerosis → hypertension, so the patient receives antihypertensive drugs "iatrogenic"

○ Most of patients know the hazards of D.M. including impotence → anxiety & depression "psychic"

❖ What are investigations and treatment of impotence?

↳ Investigations : Doppler US

↳ Treatment :

- Treatment of the cause e.g. control of DM & hypertension
- Medical treatment e.g. vasodilators e.g. papaverin
- Surgery e.g. penile prosthesis "fixed or inflatable"

General Examination Of Neurological Case

❖ Examination as general examination but stress on the following:

I. Vital signs

❖ Temperature	❖ Pulse	❖ Blood pressure	❖ Respiratory rate
See below	❖ Irregular "AF" in embolism	▪ Hypertension in stroke (thrombotic or hemorrhagic)	See below

❖ What are the Causes of fever in a neurological case?

1. Brain	2. Brain stem	3. Spinal cord	4. Peripheral nerve	5. Muscle
- Brain abscess - Meningitis - Encephalitis - Encephalomyelitis	Pontine hemorrhage	- Poliomyelitis - Transverse myelitis	Landry-Guillan – Barre syndrome	Myositis

II. General overview

A. Appearance	B. Built	C. Consciousness	C. Color	D. Decubitus	E. Expression
Asymmetrical face in facial palsy	- Under built: depression, anorexia nervosa - Over built: myxedema; pseudo-hypertrophy	DCL: in hypertensive encephalopathy	See below	Orthopnea in HF: dilated cardiomyopathy in Friedrich's ataxia or Duchenne's myopathy	- Mask face in Parkinsonism - Myasthenic face: in myasthenia

❖ What are the Causes of ... in neurological case?

❖ Jaundice	❖ Cyanosis	❖ Pallor
- Sickle cell anemia → embolic hemiplegia - Thalassemia (chronic hemolysis) → hemosiderosis in caudate lobe → chorea	Respiratory muscle paralysis leading to respiratory failure in myopathies	Subacute combined degeneration due to vitamin B12 deficiency

III. Systemic overview:

❖ Head	❖ Neck	❖ Upper limb	❖ LL
Bony bosses e.g. meningioma	- Congested Neck veins: - In heart failure as in myopathy - Carotid bruit: atheromatous plaque in embolic or thrombotic stroke or TIA	Skin: Angiomatous malformation may point to similar lesion in brain	- DVT: in prolonged recumbency of paralyzed patient - Ischemic signs in thrombotic or embolic hemiplegia

IV. Other systems except that of local exam:

❖ Cardiac examination	❖ Chest examination	❖ Abdominal exam.
As a cause: - Hemiplegia, may occur due to: - Embolic: source: MS & AF - Hemorrhage: - Severe hypertension, Intracranial hemorrhage - Thrombosis: atherosclerosis of small sized arteries e.g. cerebral & coronary arteries As a result: - Autonomic neuropathy of the heart leading to: - Postural hypotension, Painless infarction - Sudden death As an association: - Duchenne Myopathy, Friedreich's Ataxia - Rheumatic Chorea	As a cause: - TB; Pott's disease → paraplegia As a result: - Respiratory muscle paralysis leading to: - Hypostatic pneumonia - Respiratory failure	As a cause: - LCF → hepatic encephalopathy As a result: - Autonomic neuropathy of the GIT leading to: - Gastroparesis diabeticorum - Diabetic enteropathy diarrhea As association: - Wilson's syndrome (hepato-lenticular degeneration)

Local Examination Of Neurological Case

1. Mentality	2. Speech
3. Cranial nerves	4. Meningeal irritation
5. Motor system	6. Reflexes
7. Sensory system	8. Others : Cranium , neck, back, spine & Gait
9. Other systems	

1. Mentality

1. State of consciousness درجة الوعي
2. Orientation to time, place, person ؟ عارف أنا مين ؟ عارف انتہ فين ؟ عارف انا مين ؟
3. Memory : دلوقتي صبح ولا ليل ؟

a) Immediate memory	b) Recent memory	c) Remote memory
■ تذكر له بعض الأرقام ثم نطلب منه أن يقولها بنفس الترتيب	■ فطرت اي النهاردة ؟	■ نسأله عن تاريخ جوازه - آخر 3 رؤساء للبلد -

4. Affect (Mood):

Affect: is the outward expression of emotion Mood : is the patient's inner feelings

Abnormality include :

- Depression : stroke, parkinsonism
- Euphoria: disseminated sclerosis (D.S.)

5. Mentality (assessed by intelligent quotient test; IQ معدل الذكاء)

❖ لو الدكتور والمريض فهموا بعض ← average mentality

6. Behavior : السلوك

❖ طالما العيان اداك history وسمح لك بال examination يبقى ← Cooperative

❖ Comment on mentality: The patient is fully conscious, cooperative, well oriented to time, place & person, average memory, affect (mood), & mentality.

- ? What is the Glasgow coma scale? See discussion later
- ? What are the grades of disturbed consciousness?
- ? What are the types & causes of amnesia?
- ? What are the disorders in the mood & affect?
- ? What is hallucination , illusion, delusions, delirium, dementia ?

لهم إجابات الأسئلة موجودة في منهج ال Psychiatry بالتفصيل جداً مقرر عليك... ذكركم من مصدر واحد علشان تحافظ على ال Photo-graphic memory

2. Speech

■ يتم ملاحظته أثناء أخذ ال History

■ اطلب من المريض يقرأ الفاتحة أو يعد من 1 إلى 10

■ ممكن يقرأ الفاتحة كويس أوي رغم ان عنده speech defect لأنه متعود عليها , علشان كده الصح انك تخليه يقرأ حاجة مش متعود عليها (بلاد بره)

❖ The main speech disturbance include:

- A. **Aphasia**: inability to formulate speech (lesion in speech center) in absence of lesion of sense organ or mental defect & occurs in the cortical lesions affecting the dominant hemisphere.
- B. **Dysarthria** : inability to articulate speech (lesion in neuromuscular junction) with normal formulation (normal speech center)

❖ Types of dysarthria :

1. **Slurred speech** : ياكل الكلام بسرعة due to
 - Bilateral pyramidal tract lesion = pseudo bulbar palsy
 - Lesions involving the LMN of the cranial nerve concerned with speech (5, 7, 10, 12)
2. **Staccato speech** : الكلام متقطع : bilateral cerebellar lesion (hereditary ataxia, D.S.)
3. **Monotonous speech** : الكلام على وتيرة واحدة : extrapyramidal tract lesion (parkinsonism)
4. **Scanning speech** = slurred + Staccato in cerebellar lesions

3. Cranial nerve examination

CN 1 : olfactory nerve



المادة لها شروط :	(ب) العيان له شروط :
تكون معروفة للعيان زي البن أو النعناع	قافل عينه ومش شايف المادة
متيقن irritant علشان تتجنب ال 5 th CN irritation	فحص كل nostril على حده

شروط ما قبل الفحص:

المادة لها شروط :

- طريقة الفحص:
- في نفس الوقت بنفس اليد : الدكتور يغلق العينين واقل واحدة من ال nostrils
 - واليد الثانية اشمع المادة
 - ثم كرر الخطوات السابقة على ال nostril الأخرى

❖ Abnormality :

- Unilateral anosmia : Neurological; basal meningitis, Fracture skull base, Foster Kennedy syndrome
- Bilateral anosmia : local ENT causes & hysterical

CN 2 : optic nerve

1. Acuity of Vision:

A. Classically by: Snellen's chart more than 6 meters distance.

B. Bedside test: for less than 6 meters distance:

منفص عنيه الاتنين , كل عين مرة لوحدها , واطلب منه يفتح عين ويغمض التانيه

- Counting finger from 6m to 30 cm, if the patient cannot, do the next step;
- Hand movement at 30 cm distance, if the patient cannot, do the next step;
- Perception of light:
 - No perception of light means blindness.

2. Field of vision :

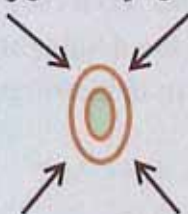
A. Classically by: Central vision (Bjerrum's screen) & peripheral vision (Perimeter)

B. Bedside test: Confrontation test:



طريقة الفحص :

- الدكتور والعيان لازم يكونوا على نفس المستوى
- المسافة ما بينهم تتراوح ما بين 60 - 100 cm
- لو الدكتور أو العيان من ذوي النظارات لازم تتشال , ليه ??? ← rim of glasses may create artificial visual field defect
- المريض يغمض عين والدكتور يغمض العين المقابلة لها
- أهم جملة لازم تقولها وعليها الدرجة كاملة : ثبت عينك في عيني ومتحركهاش ولما تشوف صباعي قولي
- الدكتور يحرك صوابه من بره لجوه بحركة سامبوكسة في أربع اتجاهات مع استخدام اليد المناسبة في فحص ال field of the vision
- على سبيل المثال عند فحص العين اليمنى للمريض - المريض يقلب عينه الشمال و الدكتور يقلب عينه اليمين :
- حرك صواب اليد اليسرى لفحص ال lateral field
- حرك صواب اليد اليمنى لفحص ال medial field
- والعكس في العين الشمال للمريض
- لما يقولك شايف , قوله صباعي بيتحرك ولا لا / ثابت ولا لا ؟
- كرر الخطوات السابقة في العين الأخرى



❖ Results :

- Normal field: if the patient notices your finger at periphery the same time as you do
- Field defect: according to the site of the lesion in optic nerve pathway e.g. optic chiasma lesion → bitemporal hemianopia
- Ophthalmoscope : for papilledema, optic atrophy
- Colour Vision by Ishihara's chart for color blindness.

Ocular cranial nerves: CN 3: oculomotor, CN 4: Trochlear, CN 6: Abducent, :

Ocular muscles:

A. Extra-ocular muscles

- A. 4 recti : superior, inferior, medial, lateral recti
- B. 2 oblique: inferior oblique, superior oblique
- C. Lid muscles :
 - levator palpebrae superioris: elevates the upper eye lid
 - Muller's muscle (smooth muscle adjoining LPS): maintains the upper eye lid elevated

B. Intra-ocular muscles

- A. Pupil:
 - Dilator pupillae
 - Constrictor pupillae
- B. Lens:
 - Ciliary muscle : responsible for light & accommodation reflex

❖ Nerve supply

- ❖ All Extra ocular muscles are supplied by the oculomotor nerve except :
 - Muller's muscle by sympathetic nerves
 - Lateral rectus by abducent nerve (LR6)
 - Superior oblique by trochlear nerve (SO4)

- Dilator pupillae : by sympathetic nerve supply
- Constrictor pupillae & Ciliary muscle: parasympathetic supply through oculomotor nerve

❖ Lesion:

A. External ophthalmoplegia

- ❖ No diplopia, **Diplopia** occurs only on passive elevation of eye lid
- ❖ **Complete ptosis** (paralysis of LPS)
- ❖ **Divergent paralytic squint**, the eye looks out & down due to unopposed action of SO & LR

B. Internal ophthalmoplegia

- Ipsilateral **Dilated fixed pupil** (Mydriasis)
- **Loss of light & accommodation reflex**

→ So CN 3 lesion : 3 D + loss of 2 reflexes + complete ptosis

❖ CN 4 lesion

- ❖ Diplopia on looking downwards
وهو نازل السلم أو يقرأ
- ❖ Limitation of eye movement on looking inwards & downwards

❖ CN 6 lesion

- ❖ Diplopia on looking outwards
- ❖ Limitation during examination of eye movement in outward direction
- ❖ Convergent paralytic squint

❖ Sympathetic lesion "Horner's syndrome"

- ❖ Unilateral partial Ptosis due to lesion in Muller's muscle
- ❖ Miosis due to lesion in dilator pupillae
- ❖ Anhidrosis, Enophthalmos
- ❖ Lost spino-ciliary reflex
- ❖ Conjunctival congestion

❖ Examination of ocular nerves : examination of:

A. Inspection

B. Power

C. Reflexes

A. Inspection

1. Ptosis

2. Squint

3. Size of pupil

4. Nystagmus

1. Ptosis :

- ❖ **Normally** : upper eye lid covers 1/6 of the cornea
- ❖ **Abnormally** Ptosis: upper eye lid covers > 1/6 of the cornea



❖ Causes :

- ❖ 3rd nerve palsy & sympathetic palsy
- ❖ Myasthenia gravis (bilateral with normal pupil + other myasthenic manifestations + diurnal variation)
- ❖ Myopathy, Motor neuron disease
- ❖ Congenital, traumatic

❖ إزاي تفرق ما بين ال 3rd nerve palsy & sympathetic palsy ؟

3rd N. palsy	Sympathetic palsy
○ Dilated fixed (mydriasis) & Divergent squint	I. ارفع الحاجب وبص على ال Pupil : Miosis, Enophthalmos of eye ball II. اعمل Thumb test : ▪ اطلب من المريض انه يقفل عينه ▪ دوس بال thumbs على ال superior orbital margins ▪ اطلب من المريض انه يحاول يفتح عينه ○ لو قدر ولو قليلا Partial ptosis ○ لو مقدرش complete ptosis

2. Nystagmus :

❖ **Definition :** involuntary rhythmic oscillatory movement of the eye ball

→ N.B.: Nystagmus is a sign not a symptom

❖ **Causes of nystagmus:**

a) Physiological

b) Pathological:

▪ Central causes: brain stem, cerebellum ▪ Peripheral causes : retinal, vestibular



Nystagmoid feature اسمه ناستاغمويد , وده اللي عادي ,

طريقة الفحص :

1. خط صباغك بزاوية 30 درجة على العين لبره على مسافة 30 سم

2. ملاحظة: لو خطيت صباغك بزاوية 180 درجة على العين ,

عضلات العين تتعب أوي ويبان nystagmus في أي شخص عادي ,

3. خليه بيص عليه لمدة حوالي 30 ثانية

4. يتم الفحص مرة للعين اليمين ومرة للعين الشمال

5. يتم الفحص في كل عين : مرة لفوق ويثبت عينه ومرة لتحت ويثبت عينه

وبعد كده لو فيه nystagmus حدد ال :

❖ **Comment points :**

a) Type : Spontaneous or with fixation

b) Plane : horizontal or vertical or rotatory

c) Phase :

• Monophasic (pendular راحة وجاية بنفس السرعة) or

• Biphasic (jerky واحد بطيئ والتاني سريع كأنه بينط بينط)

d) Direction of nystagmus :

• If Biphasic : Direction of nystagmus is that of rapid phase

e) Laterality :

• Unilateral (dissociated) or bilateral (associated)

❖ **Characters of cerebellar nystagmus :**

a) Type : with fixation

b) Plane : horizontal

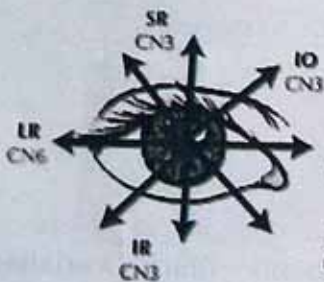
c) Phase: biphasic

d) Direction : towards the fixation point

e) Laterality : bilateral (associated)

B. Power

1. Each eye separate :



طريقة الفحص :

قواعد الفحص :

• تكلم العين وتشاور له الحركات مع بعض

• العين يثبت رقبته ويقفل عينه بإيده

• ثبت رأسه بإيدك

• اطلب منه انه يحرك عينه مش رأسه

ثم :

→ CN 6	lateral rectus ← عينك لبره
→ CN 3	وهي بره فوق SR ثم تحت IR
	medial rectus ← عينك لجوه
→ CN 4	وهي جوه لفوق IO
	ثم تحت SO

إزاي تعرف ان فيه weakness ولا مافيش ؟

• عن طريق ال range of movements

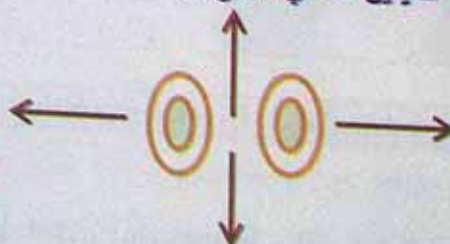
• If weakness → rim of sclera

2. Both eye together (conjugate eye movement):



• لا تخبر الا اذا كانت كل عين حركتها سليمة لوحدها والا أصبح عبثاً

• تطبيق القواعد: ثم فحص حركة العينين معا في الأربع إتجاهات :



(1) عينيك الاتنين فوق

(2) عينيك الاتنين تحت

(3) عينيك الاتنين يمين

(4) عينيك الاتنين شمال

Lesion in :

- Cortical conjugate center (area 8) → lost conjugate movement to hemiplegic side
- Pontine conjugate center → lost conjugate movement to opposite side of hemiplegia
- Medial longitudinal bundle (M.L.B.) → Ophthalmoplegia internuclearis : Pathognomonic to Disseminated sclerosis :

→ on looking to one side there is :

- Nystagmus of abducting eye
- Weakness or paralysis in adducting eye



Monocular nystagmus



Paralysis

ما نوع ال conjugate eye movements التي اختلقتها ؟

Voluntary conjugate eye movements

هل فيه أنواع تقيه ؟

Reflex conjugate eye movements

هل إمتى ؟

Oculo-cephalic reflex → negative reflex in comatose patient's means that brainstem is not intact

C. Reflexes

Reflex	A. Accommodation (near) reflex: → Normal pupil is RRR (round, regular, reactive to light & accommodation)	B. Pupillary Light reflex:
Technique	هات صباغك من بعيد وقوله تابع صباغي لحد حوالي 20 سم أو (عند الطلب) : قول للعيان ببص على مكان بعيد ويركز في نقطة فيه وفجأة حط صباغك قدامه وقوله ببص على صباغي	- افصل ما بين العينين بأن تضع يدك حاجز - حط الضوء على العين من الجنب (ويفضل والنور مقفول) - راقب سرعة ضيق الحدقة (Direct) - شيل الضوء - بص على العين الثانية الأولى ثم ضع الضوء مرة أخرى - راقب سرعة ضيق الحدقة في العين الثانية (Indirect)
Stimulus :	Near object	Light to the eye
Pathway	Optic nerve (afferent) → optic chiasma → Optic tract → Lateral Geniculate Body → visual area (17) in occipital lobe (center) → area 8 → E.W. nucleus both sides in midbrain → both 3rd nerve (efferent) → 3 muscles (constrictor pupillae, medial recti & ciliary muscles)	Optic nerve (afferent) → optic chiasma → Optic tract → pretectal nucleus in the midbrain (center) decussating around aqueduct of sylvius → Edinger Westphal nucleus both sides → both 3rd nerve (efferent) → ciliary ganglia → short ciliary nerves → constrictor pupillae muscles
Response :	- Bilateral Constriction of both pupil (both miosis) by constrictor pupillae muscles. - Bilateral convergence (both MR) - Bilateral contraction of ciliary muscles → convex lens → focusing	Ipsilateral (direct) & contralateral (Consensual) constriction of pupils
Lesion:	- In optic nerve atrophy : absent reflex - In 3rd Nerve palsy : ipsilateral loss of response	- In optic nerve atrophy : absent direct & consensual LR - In 3rd Nerve palsy : ipsilateral absent direct & present consensual LR

C. Cilio-spinal reflex :

- Technique: pinching of skin on one side of neck
- Normal Response : dilatation of ipsilateral pupil.
- Abnormally absent in: sympathetic lesion (Horner's syndrome)

❖ What are the causes of miosis & mydriasis?

❖ Causes of mydriasis	❖ Causes of miosis
- Parasympathetic (III CN) lesion - Adie's pupil - Optic nerve atrophy (blindness) - Drugs: atropine & cocaine - Physiological : poor light & frightened person	- Sympathetic lesion (Horner syndrome) - Argll-Robertson pupil - Potome hemorrhage - Drugs : morphine, pilocarpine - Physiological : bright light & sleep

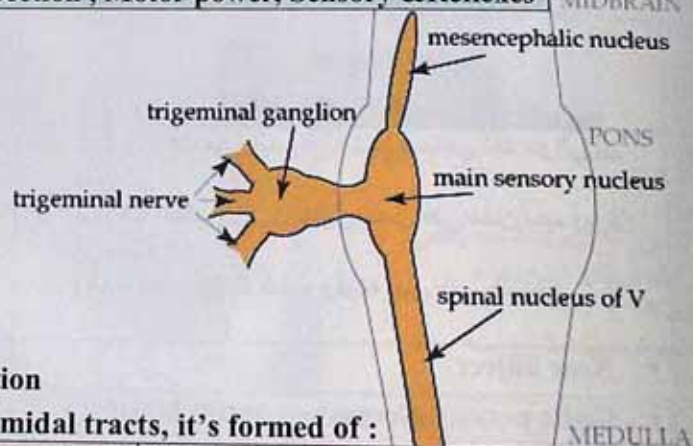
❖ What is the difference between Argyll-Robertson pupil & Holme's Adies pupil?

	Argyll-Robertson pupil	Holme's Adies myotonic pupil
Causes	- Lesion in prei-aqueductal region of the midbrain : - Neurosyphilis, Syringobulbia - DS, DM , Chronic alcoholism - Encephalitis	It is a benign heredofamilial , usually unilateral, of acute onset, occurring in females
Pupil	Miotic, irregular, eccentric pupil	Dilated pupil
Light reflex	Loss of light reflex	Loss of light reflex
Accommodation reflex	preserved accommodation	Very slow reaction to accommodation On relaxation from accommodation , pupil dilates very slowly (tonic pupil)
Others	Sluggish response to cilio-spinal reflex & to mydriatics	It may be associated with lost ankle reflex

❖ Examination of CN :

CN 5	CN 7	CN 9,10
Examination by Inspection , Motor power, Sensory & Reflexes		

CN5; Trigeminal nerve:



❖ Hint of anatomy :

- It is mixed nerve "motor & sensory"
- Mainly sensory to face
- Supply motor fibers for muscles of mastication
- Its nucleus takes bilateral supply from pyramidal tracts, it's formed of :

Nucleus	Site	Sensation
Mesencephalic nucleus	Midbrain	Proprioceptive
Main sensory nucleus	Pons	Touch
Spinal nucleus	Medulla	Pain & temperature

→ In the Spinal nucleus:

- The face is represented upside down i.e. Ophthalmic fibers end in lower part of the nucleus and mandibular fibers in the upper part of the nucleus
- The peripheral part of the face is represented in the inner part of the nucleus and the central part of the face is represented in the outer part of the nucleus

1. Inspection for hollowing:



شوف هل في تجويف :
temporalis muscle ← zygoma فوق ال
masseter muscle ← zygoma تحت ال
شوف ال symmetry , هل الناحيتين زي بعض ولا لا

Motor Power:

A. Temporalis muscle



■ حط إيدك على العضلة وقول للعيان جز على سنائك وسيب

B. Masseter muscle



■ إمسك العضلة وحس ال bulk وقول للعيان جز على سنائك وسيب

C. Pterygoid muscles:

- كل Pterygoid muscle تزق الفك للناحية المقابلة
- العضلتين معا تنزل الفك لتحت مباشرة

1. قوله افتح برك وشوف حركة الفك :

- اللون الأسود : الدكتور
- اللون الأزرق : المريض



لو نزل في النص يبقى حاجه من اثنين :

- الناحيتين ضعيفة
- الناحيتين سليمة

إزاي تفرق ← against resistance

■ حط إيدك الشمال فوق راسه علشان تثبتها فتتضمن انه يستخدم فكه مش راسه

■ حط إيدك اليمين تحت فكه واضغط لفوق جامد وقوله افتح برك

→ In bilateral weakness or paralysis : inability to open mouth against resistance

لو حود على ناحية : تستفيد ان :

- ان الفك ببشاور على الناحية الضعيفة (فكسان عيان)

○ Unilateral LMNL

2. ممكن تختبر كل Pterygoid muscle على حده (عند الطلب) :

- حط إيدك الشمال فوق راسه علشان تثبتها فتتضمن انه يستخدم فكه مش راسه
- حط قبضة إيدك اليمين على جانب الفك وقوله حاول تزق إيدي (لاحظ انك بتختبر العضلة بتاعة الناحية المقابلة)

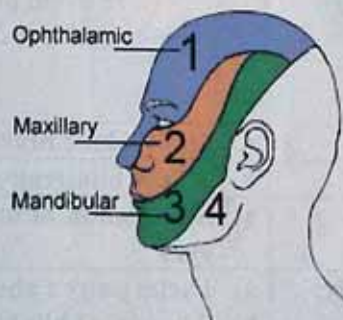
لو انتة كده بتفحص اي جزء من ال Pterygoid muscle ؟ ← بفحص ال lateral part

Sensation :

❖ Sensation from the face are carried through trigeminal branches :

1. Ophthalmic branch: skin over forehead
2. Maxillary branch : skin over cheeks
3. Mandibular branch : skin over lower jaw :

→ Except the skin over the angle of mandible which is supplied by C2



لو طبيب ليه مش C1 ؟

❖ C1 has no skin supply it's purely motor root, share with the accessory nerve.

N.B.:

- Trigeminal nerve is responsible for GENERAL sensation of anterior 2/3 of tongue



- طريقة الفحص: إبدأ بالسليم وداوما اسأله حاسس هنا زي هنا ؟
 إستخدام قطنه لفحص ال pain , إستخدام قطنه لفحص ال touch
- (1) اطلب من العيان أنه يغمض عينيه
 - (2) مقارنة الإحساس في الناحيتين : ناحية اليمين وناحية الشمال في الثلاث مناطق (1 , 2 , 3)
 - (3) مقارنة الإحساس بين أربع مناطق (1 , 2 , 3 , 4) على ناحية واحدة (تفرق ما بين ال organic وال hysterical)
 - (4) مقارنة الإحساس في ناحية واحدة بين :
 - (5) مقارنة الإحساس في ناحية واحدة بين :
- حته central ← جنب الأنف
 - وحته peripheral ← جنب الأذن

❖ Abnormality :

- 1) Unilateral lesion of nerve: loss of sensation over same side of face sparing angle of mandible
- 2) Dissociative sensory loss : lesions in medulla
- 3) Lower face sensory loss with normal upper face: lesion in pons
- 4) Loss of sensation in the center of face & preserved peripheral in Tabes dorsalis (peripheral nuclear lesion)
- 5) Loss of sensation in the periphery of face & preserved central sensation in syringobulbia (central nuclear lesion)

4. Reflexes :

	1) Corneal (conjunctival) reflex :	2) Jaw reflex "temporalis masseter reflex"
		
▪ Stimulus & technique:	▪ قول للعيان يبص الناحية الثانية , ليه ؟ ▪ to avoid photic stimulation ← ▪ إعمل سن للقطنة وتعالى من جنب العيان والمس عينه بالقطنة	▪ قول للعيان افتح بفتحك نص ففتح ▪ حط صباغك ال index تحت الشفة السفلى في تجويف الذقن ▪ اخبط صباغك بال hammer من فوق لتحت ▪ في الطبيعي ما يحصلش أي حاجة
▪ Afferent	▪ Ophthalmic branch of 5 th CN	▪ 5 th CN
▪ Efferent :	▪ 7 th CN bilaterally	▪ 5 th CN
▪ Response: Normally	▪ Stimulation of one eye → blinking of both eyes	▪ Normally absent
▪ Abnormally:	a) Facial palsy : absent blinking in same eye b) Absence of blinking in both eye is seen in : ↳ trigeminal affection of stimulated side ↳ Bilateral facial palsy ↳ Organic type of coma	• If present (closure of mouth) in bilateral UMNL above the pons e.g. in pseudobulbar palsy • Jaw reflex is one of the leveling reflex in quadriplegia

3) Palatal reflex : see later

CN7; Facial nerve:

Hint of anatomy :

- It is a mixed cranial nerve " sensory & motor"
- Mainly motor " supplies facial muscles"
- Carry sensory fibers to anterior 2/3 of the tongue
- It's nucleus present in pons:

The upper part of the facial nucleus:

Receives bilateral pyramidal supply from both

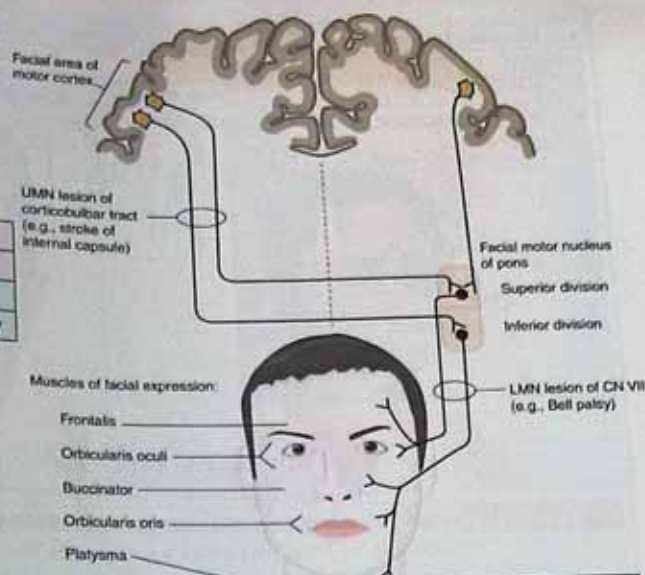
The lower part of the facial nucleus:

Unilateral pyramidal supply from the contralateral side only

Possibilities of lesions:

- Unilateral UMNL or Unilateral LMNL
- Bilateral UMNL or bilateral LMNL

1. Inspection for :



A. Upper face		B. Lower face:	
• Frontalis muscle	• Orbicularis oculi	• Buccinator	• Retractor anguli
○ Absence of corrugation of the forehead	○ Infrequent blinking & epiphora	○ Salivary dribbling from the diseased side	○ Absence of nasolabial fold in the diseased side
			○ Deviation of mouth to healthy side

2. Motor power:

A. Upper face	
• Frontalis muscle	• Orbicularis oculi
Without resistance:	
○ قول للعيان: ارفع حواجبك بص للسقف	○ زر على عينك جامد
○ شوف الحاجبين ارتفعوا زي بعض ولا لا	
○ التجاعيد ظهرت على الناحيتين ولا لا	
With resistance:	



○ اطلب من المريض انه يرفع حواجبه وننته ضاغط بايديك جامد

○ زر على عينك جامد ومتخلنيش افتحهم وحاول تفتحها للعيان



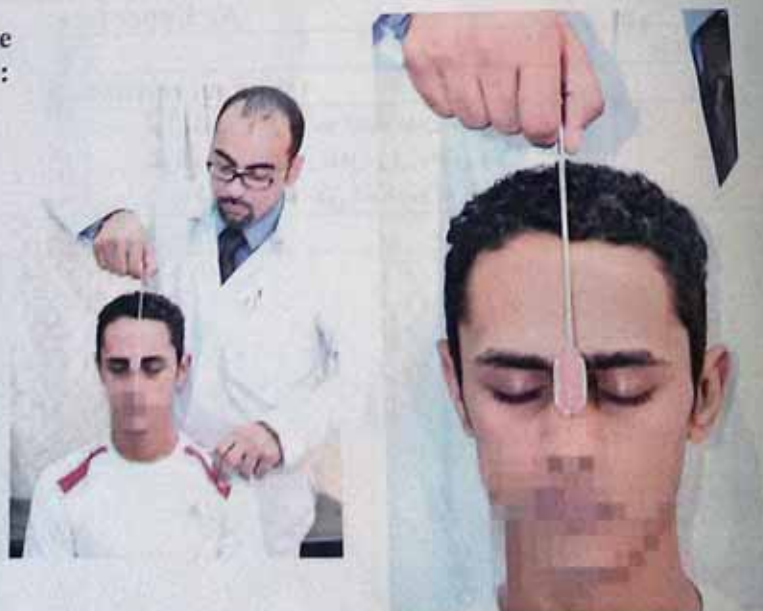
3. Sensation: TASTE sensation of anterior 2/3 of tongue by corda tympani



1) ممنوع العيان يتكلم أثناء الفحص	2) العيان يغمض عينيه	3) شد اللسان ليره وتشغله بشئ
4) القسم اللسان يمين وشمال	5) ضع المادة على الجزء الذي تريد اختباره	6) يكتب أو يشار به لو حاسس بالطعم

4. Reflexes :

- 1) Corneal (conjunctival) reflex : see before
- 2) Glabellar reflex "nasopalpebral reflex" :



• Stimulus :

قل للعيان بص للأرض أو أقل عيناك نص قفله أو سبل عيناك
امسك ال hammer من أعلى بحيث يتدلى منك وإخبط على قاعدة الأنف (root of the nose = glabella)

▪ Afferent : 7th CN ▪ Efferent : 7th CN

- Response: Normally: blinking of both eyes 3-5 times (contraction of orbicularis oculi), then stops due to habituation.

→ Abnormally:

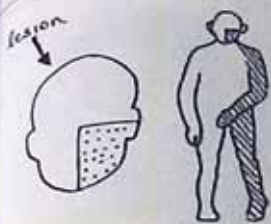
- a) Continuous blinking with taps as long as stimulus is applied in Parkinsonism (Myerson's sign)

b) Exaggerated in bilateral UMNL

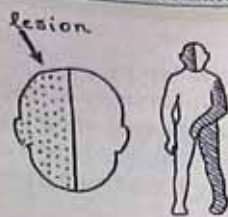
c) Lost in bilateral LMNL

Difference between UMN & LMN of facial paralysis :

❖ Unilateral UMN of facial nerve



❖ Unilateral LMN of facial nerve



- Affecting Δ tract above facial motor nucleus in the pons.
- Paralysis of the muscles of lower half of the face on the opposite side of the lesion (supplied from the opposite Δ tract only):
 1. Obliteration of the naso-labial fold
 2. Drooping of the angle of the mouth with dripping of saliva
 3. Accumulation of food behind cheek
 4. Inability to blow the cheek
 5. Inability to show teeth properly
- Paralysis involves the voluntary movement; it spares the emotional & associative movements (which are supplied by extra Δ fibers).
 - لو العيان ضحك بمزاجه ← بقة يتعوج
 - لو ضحكته من قلبه ← ما يتعوجش
- Paralysis is associated with hypertonia & hyperreflexia.
- There is associated hemiplegia on the **same** side of the facial paralysis.

- Affecting the facial motor nucleus or the nerve itself.
- Paralysis of the muscles of the upper & lower halves of the face on the same side of the lesion.
- In addition; there are:
 6. Inability to raise eyebrows with absence of wrinkles of forehead
 7. Inability to close the eye;
 - Bell's phenomenon: on firm closure of eyes $\rightarrow$ up rolling of eye balls (used to differentiate organic & hysterical facial palsy)
- Paralysis affects voluntary, emotional & associative movements.
- Paralysis is associated with hypotonia & hyporeflexia.
- If there is hemiplegia, it is on the **opposite** side of the facial paralysis (**crossed** hemiplegia).

❖ Bilateral UMN OR bilateral LMN of facial nerve: taking all the face ;

❖ Causes of bilateral facial palsy: Mnemonic : **Bull DOG LIMPS**

- **Bull** = Bulbar palsy, Basilar skull fracture and Botulism
- **D** = Diabetes Mellitus
- **O** = Osteopetrosis
- **G** = Guillain-Barre syndrome
- **L** = Leukemia, Leprosy and Lyme disease
- **I** = Isoniazid and Infectious mononucleosis
- **M** = Myasthenia gravis, Myotonia dystrophica, Meningitis, Moebius syndrome and Malaria
- **P** = Periarthritis nodosa, Poliomyelitis, Porphyrias and Postvaccination neuropathy
- **S** = Syphilis and Sarcoidosis (Heerfordt syndrome)



• Bilateral UMN of facial nerve:

- Exaggerated glabellar reflex
- Present Jaw reflex

$\rightarrow$ Differentiation by :

- Bilateral LMN of facial nerve
- Lost glabellar reflex
- Absent Jaw reflex

Bulbar CN: CN 9; Glossopharyngeal, CN10; vagus, CN11; cranial part of accessory nerve

❖ Introduction:

- They supply palate, pharynx & larynx
- Lesion in them called : palate-pharyngo-laryngeal paralysis = bulbar palsy
- Pyramidal tract supplies cranial nuclei of both sides of bulbar nerve so :
 - Unilateral lesion i.e. unilateral bulbar palsy can occur but is always of = LMNL
 - While bilateral lesion may be UMNL or LMNL



قول العيان يفتح بقة

نزل اللسان ب torch وبص على ال base of the uvula (مش على ال tip لأن ال tip أصلا)

1. Inspection & Motor Power :

- Uvular deviation to healthy side : contralateral unilateral LMNL
- IF central uvula : خلي العيان يقول آآآه
 - Symmetrical elevation of soft palate : Normal
 - Non mobile uvula and palate : Bilateral palsy either in bilateral UMNL or bilateral LMNL

2. Sensation :

- GENERAL & TASTE sensation of posterior 1/3 of the tongue
- How to test taste sensation in posterior 1/3 of tongue?
- It's tested by using electric current (4 mill-ampere) → metallic taste

3. Reflexes :

	1) Palatal reflex	2) Pharyngeal reflex "gag reflex"
		
Stimulus	2 tongue depressor; one depresses the tongue & the other touching the soft palate	2 tongue depressor; one depresses the tongue & the other touching the pharynx (posterior pharyngeal wall)
Afferent	5 th CN	9 th CN
Efferent	10 th CN	10 th CN
Response : Normally	Elevation of soft palate slowly	Gag; if the patient annoyed, feel the desire to vomit
Abnormally :	- No elevation of soft palate : bilateral LMNL - Exaggerated elevation of soft palate : bilateral UMNL	- No annoyance: bilateral LMNL - Exaggerated response : the patient vomits: bilateral UMNL

إزاي نقدر نفحص ال vagus nerve لوحده ؟



Indirect laryngoscope to observe position & movements of vocal cords

هل ممكن ال glossopharyngeal nerve يتصاب لوحده ؟

أه ممكن بس rare جدا زي مثلا حالة neuroma of glossopharyngeal nerve

ليه rare ؟

Vernet's syndrome لأنهم طالعين من ال jugular foramen ويكون اسمها

إزاي نفرق ما بين ال Bilateral LMNL وال Bilateral UMNL ؟

	Bilateral UMNL = pseudo-bulbar palsy	Bilateral LMNL = true-bulbar palsy
	→ Differentiation by	
5 th CN	Exaggerated jaw reflex	Absent
7 th CN	Exaggerated glabellar reflex	Lost glabellar reflex
9 th & 10 th CN	Exaggerated palatal & pharyngeal reflex	Lost palatal & pharyngeal reflex
12 th CN	Spastic tongue	Flaccid, atrophic tongue
	No fasciculation	Fasciculation

CN 8; Cochleo-Vestibular nerve

A. Cochlear part : test for acuity of hearing using :

1. Watch test



2. Rinne's test



3. Weber's test



- The acuity of the patient's hearing is compared to that of the examiner's
- If there is diminution of patient acuity for hearing do the following test

❖ Technique :

- Using vibrating tuning fork, compare air conduction (in front of ear) with bone conduction (on mastoid process)

- Place tuning fork in the middle of head

❖ Normally :

- Air conduction is better than bone conduction

- The vibrations are heard in the middle of forehead

❖ Nerve deafness :

- Both air and bone conductions are diminished

- The vibrations are heard in the normal ear

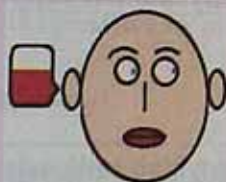
❖ Conductive deafness :

- Bone is better than air

- The vibrations are heard in the affected ear

B. Vestibular part :

1. Vestibulo-ocular reflex or caloric reflex test:



2. Oculo-cephalic reflex, or doll's eyes reflex:



❖ Aim :

- Assess function of internal ear

- Bed side test to evaluate vestibular functions
- Differentiate between causes of nystagmus

❖ Technique :

- Patient lie supine, his head flexed 30° so that the lateral semicircular canal is vertical $\rightarrow$ generate a maximal response
- Each ear is douched with water for 40 seconds
- 3°C (7° below normal)
- 4°C (7° above normal)

- Patient lies on his back, with his shoulder at the end of the bed. His head, projecting beyond bed, is supported by examiner hand
- Head is then fully extended and turned to one side
- Patient's eye should remain open
- After a short interval, test should be repeated with head extended and rotated to other side

❖ Results :

❖ Normally :

- A positive response occurs when the eyes conjugately deviate in the direction opposite to the head's rotation; thus indicating that the brainstem (CN3, 6, 8) is intact.

❖ Abnormally :

- $\rightarrow$ Nystagmus (may appear within 10 seconds) :
 - If it remains < 1 minute : lesion in inner ear (same side)
 - If it remains > 1 minute : lesion in 8th nerve or cerebellum

- If the water is warm $\rightarrow$ Both eyes will turn toward the contralateral ear, with horizontal nystagmus to the ipsilateral ear.
- If the water is cold, $\rightarrow$ both eyes then turn toward the ipsilateral ear, with horizontal nystagmus to the contralateral ear.
- Absent reactive eye movement $\rightarrow$ vestibular weakness of the side being stimulated
- If the eye movement & nystagmus are absent
- Brainstem reflexes are damaged

CN 11; accessory nerve

❖ Has 2 parts:

- 1) Cranial accessory part: distributed with vagus nerve
- 2) Spinal accessory part:

• Sternomastoid :

• Trapezius:

1) Inspection :

- Head tilt to affected side

- Shoulder depression

2) Motor power:

exposed تكون muscle
من وراء العيان
تواجه العضلة
Against resistance



❖ من قدام العيان :

1. كل عضلة لوحدها: (كل عضلة تزق الرقبة الناحية العكسية)
○ حط إيدك على جنب فكك وقوله زق إيدي وحس العضلة الناحية الثانية
○ حط إيدك الثانية بنفس الطريقة وحس العضلة الأخرى
2. العضلتين مع بعض: (العضلتين مع بعض يزقوا الرقبة لتحت)
○ حط إيدك تحت ذقنه وقوله زق إيدي وشوفهم وحسهم الاتنين

من وراء العيان:
خط إيدك الإثنين على كتف العيان وزق وقوله ارفع كتافك فوق

- N.B.: sternomastoids & trapezius are proximal muscles, so receive pyramidal supply from both sides

CN 12; hypoglossal nerve

1. Inspection :

- Fasciculations while tongue inside the mouth.

- يمنع الحكم عليها وال tongue بره الفم ,
ليه ؟ ← physiological fasciculations
○ تطلب من العيان يفتح بقة
○ تستخدم torch وتشوف ال fasciculations

- Wasting which indicating LMNL

ال m.m. ال tongue بقى على شكل folds أكن هدمومه وسعت عليه



2. Motor :

- A. Without resistance: إطلب من العيان أنه يطلع لسانه لبره



- If deviated → it towards the paralyzed muscles فكسان عيان, unilateral paralysis, either UMNL or LMNL

UMNL

- Unilateral: deviation to opposite side of lesion
- Bilateral: inability to protrude tongue (spastic tongue)
- In both cases there is no wasting nor fasciculation

LMNL:

- Unilateral : deviation to same side of lesion
- Bilateral : inability to protrude tongue
- In both cases there is wasting & fasciculation, tongue is sickle, corrugated & wasted

B. With resistance:



تزق من بره بصباك ولسان العيان يزق إيدك من جوه

N.B.:

They have bilateral supply for CN 12 (50 % of people)
May be affected then recovered (DD by history)

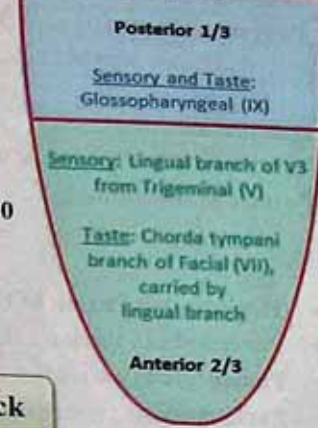
What are abnormal movements of tongue?

- Fasciculations : nuclear lesion in motor neuron disease & syringobulbia
- Involuntary movements as in chorea
- Dimpling of the tongue on tapping it, in cases of myotonia.
- Tremors in parkinsonism

What is the Nerve supply of the tongue?

- Motor : hypoglossal nerve except palatoglossus by pharyngeal branch of CN10
- Sensory:
 - Anterior 2/3 :
 - Taste sensation : 7th CN
 - General sensation : 5th CN
 - Posterior 1/3 : taste & general sensation : 9th CN

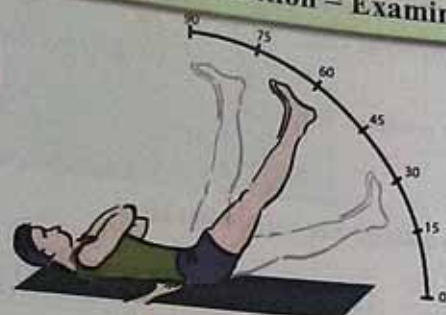
Motor: Hypoglossal (XII), except Palatoglossus: Pharyngeal branch of Vagus (X)



4. Meningeal Irritation = Examination of neck



Opithotonus



Lassegue's sign



Kernig's sign

❖ **Meningeal irritation signs** : positive in meningitis, sciatica, subarachnoid hemorrhage

1. Neck rigidity
2. Opithotonus (high arched back)
3. Lassegue's sign :
 - Raise the leg of a supine patient by flexion of hip (while the knee is extended),
 - Normally it can be raised to 90° without discomfort.
 - Abnormally : it is painful & limited before 90°
4. Kernig's sign :
 - Flex hip & knee at 90° in supine patient, then extend the knee slow
 - Abnormally : extension is limited & painful
5. Brudzinski's sign:
 - Neck sign : passive flexion of neck leads to flexion of both knees and hips
 - Leg sign : passive flexion of one hip leads to flexion of the other hip and knee



Neck sign

5. Examination of motor system

1. Inspection of the muscle state	2. Palpation for the muscle tone	3. Percussion for fasciculation & myotonia	4. Power	5. Co-ordination
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❖ قواعد الفحص:

1. Expose four limbs	2. نظم نفسك			
	a) Start by healthy side, then diseased side; compare both	b) UL then LL	c) Distal then proximal	c) Use your both hands

❖ شروط خاصة حسب الاختبار

1. Inspection:

A. **Abnormal Involuntary Movements** e.g. athetosis, pathological fasciculations, chorea, dystonia & tremors.

1. Describe: see details later in Parkinsonism case.

1. Pattern	2. Static or kinetic	3. Slow or rapid	4. Regular or irregular	5. Fine or flappy (coarse) tremors	6. Factors that increase & decrease the movement
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2. Distribution :

- UL or LL or both, head & neck, Unilateral or bilateral, Distal or proximal

B. **Posture of the Patient:**

- Hemiplegia : flexion of UL & Extension of LL
- Paraplegia in flexion : flexion of both LL
- Paraplegia in extension : extension of both LL
- Parkinsonism : generalized flexion attitude (Gorilla like ما تقولش)
- LMNL (Peripheral neuropathy) : foot or wrist drop



يحكم عليه مرتين , مرة والعيان واقف , ومرة وهو نايم
الجسم يأخذ شكل العضلات اللي ال tone فيها عالي



لو ما فيش تقول ← no specific posture

C. **Circumference of the muscle = state of the muscle:**

1. Normal

2. Wasting: flatness or hollowness of muscles:

? What are the causes of muscle wasting? See discussion later.

A. Unilateral:



B. Bilateral:

قارن التاحيتين ببعض في نفس المكان وقسهم بالمأزورة (إبدأ بالناحية السليمة)

قارن أجزاء ال limb ببعضها مثلا مقارنة ال thigh وال leg في ال limb الواحد

→ Wasting landmarks::

✎ Upper limbs:

✎ Start distally at interossei, thenar, hypothenar leading to prominent metacarpal bones & tendons

✎ Proximal: flat shoulder

✎ Lower limbs:

✎ Start distally at intrinsic foot muscle and interossei leads to pes cavus and prominent metatarsal bones & tendons

✎ Proximal : lateral aspect of the leg & medial aspect of thigh (vastus medialis)

3. Hypertrophied:

- True hypertrophy : increase the power

- False or Pseudohypertrophy : decrease the power

? What are the causes of Pseudohypertrophy? See discussion later.



Pseudohypertrophy



Trophic changes



Pes Planus

D. **Dysplastic (Trophic) changes:**

❖ Fall of hair, brittle nails, trophic ulcers, thin skin, loss of SC fat & Charcot's joint

? What are the diseases with marked tropic changes? See discussion later.

E. **Deformity in the skeletal system:**

❖ Abnormal position (claw hand, dropped foot)

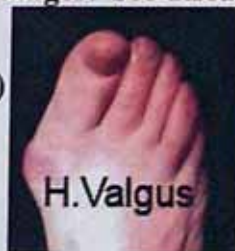
❖ Pes cavus (high arched foot)

❖ Pes plannus (flat foot)

❖ Hallux valgus or varum

❖ Hammer toes & Talipes equinovarus

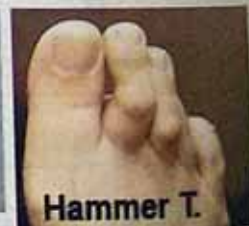
? What are the causes of pes cavus? See discussion later.



H. Valgus



H. varum



Hammer T.



Pes cavus

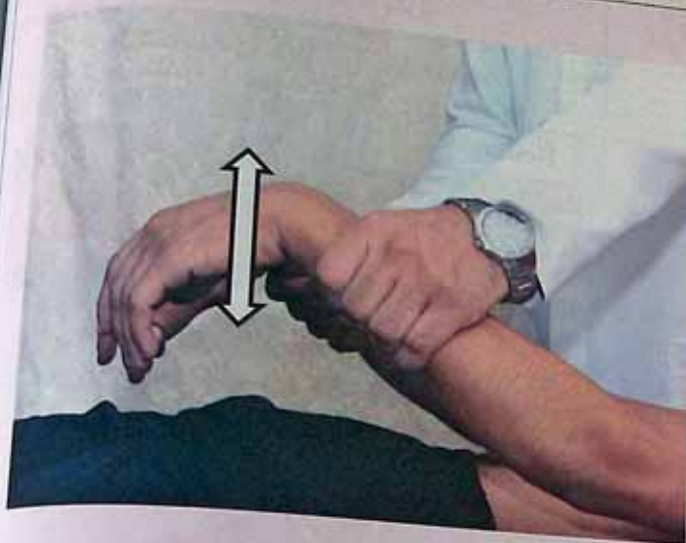
2. Palpation of muscle tone:

❦ شرط خاص هنا ← لازم تقول للعيان سيب نفسك

A. In Upper limbs

1. Wrists by Shaking method

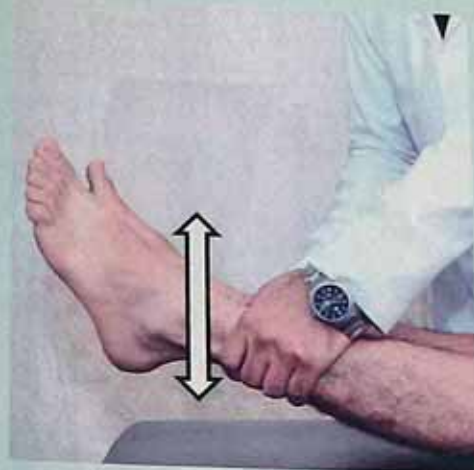
(اليمين باليمين واليسار باليسار)



B. In lower Limbs

1) Ankles by Shaking method

❦ اليد اليمين تمسك اليد أو القدم اليمين , اليد اليسار تمسك اليد أو القدم اليسار
❦ يتم فحص التلحيبتين مع بعض
❦ تهز من فوق لتحت أو من الجنب للجنب

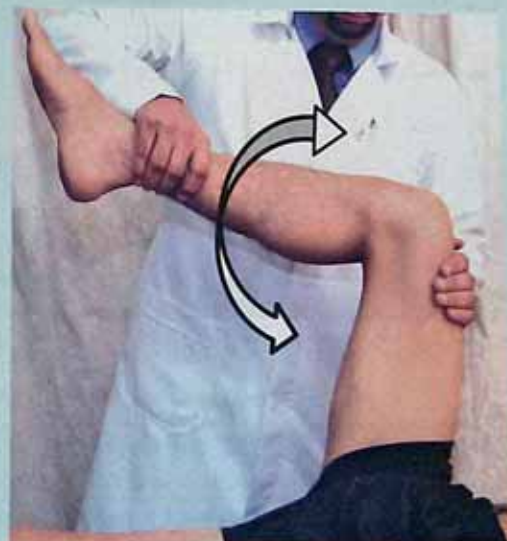


2. Elbows by Passive flexion & extension

2) Knees by Passive flexion & extension

- إيدك الشمال تثبت المفصل اللي قبل الذي تختبره
- إيدك اليمين تنثني وتفرد ال limb
- لو الحالة hypertonia :

تلي لو ال tone عالي في الأول ويسيب في الآخر ← spasticity (clasp Knife)
تلي لو ال tone عالي من الأول للآخر ← rigidity : لو معهاش tremors اسمها lead pipe , لو معها tremors اسمها Cog wheel



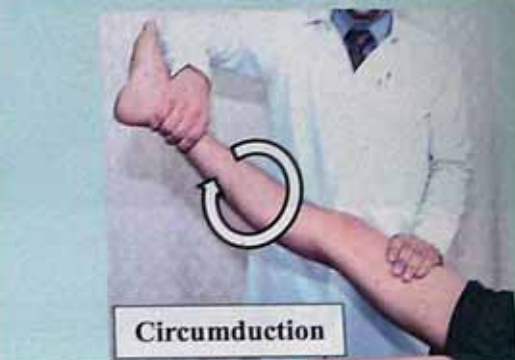
3) Shoulder: Gower's test

- المريض جالس والدكتور وراءه
- يستخدم إيديك اليمين
- حط إيديك تحت باطنه في الناحيتين معا
- واخطف الكتف لفوق
- النتيجة:
- الكتف يطلع وينزل طبيعي ← normotonia
- الكتف كانه هيتخلع ← hypotonia
- الكتف ناشف ← hypertonia



4) Hip :

- خلي العيان بفرد رجله على الآخر
- ضع إيديك اليمين فوق ال ankle وإيديك الشمال فوق ال knee
- ثم:
- مخرج رجله رايح جاي
- A. Rolling technique:
- B. Circumduction:
- ارفع رجل العيان ولغها في حركة دائرية
- النتيجة:
- الرجل تلف معك زي العجلة ← hypotonia
- الرجل مخشبة ← hypertonia



5) Both Hip & Knee by Passive elevation: طريقة الخطف

- حط إيديك تحت distal thigh ورجله مفروده على السرير واخطف لفوق وسيب
- مميزات طريقة الخطف : تختبر ال tone في ال knee وال hip
- عيوب طريقة الخطف : متحددش أنواع ال hypertonia
- النتيجة :



a) Normal

b) Hypertonia

c) Hypotonia



○ ركبته تنثني وكعب رجله ميترفعش

○ رجله كلها تطلع حتة واحدة زي المسطرة

○ ركبته تنثني ورجله تطلع لبره
○ If hypotonia is bilateral → frog position

- When you are examining the tone compare it in flexors or extensors
- If you are flexing the joint : you are examining the extensors
- If you are extending the joint : you are examining the flexors
- ❖ Results :
- a) Minimal resistance : normal tone
- b) No resistance : hypotonia (flaccidity)
- c) Marked resistance : hypertonia either:
- Clasp knife spasticity = pyramidal tract lesion or
- Rigidity = extrapyramidal; lead pipe or {cog wheel interrupted by tremors}

- ❖ What are the causes of hypotonia & hypertonia? See discussion later.
- ❖ What are other Gower's you know? See discussion later.

3. Percussion:

1) Fasciculations:

Definition : Fasciculation means spontaneous contraction of GROUP of muscle, it's visible & even palpable

N.B.: Fibrillation means spontaneous contraction of SINGLE muscle fiber, it's hardly visible except in tongue

Types:

	Physiological fasciculation	Pathological fasciculation
Cause	Anxiety, fatigue, coffee, smoking	Irritation of AHCs
Type	Coarse	Fine
Wasting	Absent	Present
EMG	Normal	Giant potentials

② **Technique :** All limbs percussed by finger flickering or by light percussion by hammer

2) Myotonia :

② **Definition :** delayed relaxation of skeletal muscle after muscle contraction (myotonic phenomena)

How to examine:

a. Mechanical myotonia: by percussion by hammer on:

- Tap of thenar eminence → adduction of thumb with delayed abduction
- Tap of the tongue → dimple formation



b. Voluntary myotonia :

- Ask the patient to catch anything or shake hand or clench fist → patient is unable to open hand except very late

c. Electrical myotonia:

- 2-3 milliampere are enough to produce muscle contraction; hyperexcitable (normally 6 milliampere is needed)

Types:

A. Myotonia congenita (hypertrophica)	B. Myotonia atrophica	C. Myotonia acquisita	D. Myotonia paradoxa
---------------------------------------	-----------------------	-----------------------	----------------------

4. Muscle Power:

❖ شروط خاصة بهذا الاختبار:

1. نظم نفسك في كل مفصل في الحركات بهذا الترتيب :

- Flexion → extension
- Abduction → adduction
- External rotation → internal rotation

2. اعمل الاختبارات مرة without resistance ومرة against resistance :

(أ) Without resistance :

- ادي العيان الأمر وأنت يتمثل الحركة بإيدك

(ب) Against resistance :

- إيدك اليمين تعمل resistance وإيدك الشمال تثبت المفصل اللي قبل الذي تختبره وقول للعيان قاومني

3. الحركة اللي العيان معرفش يعملها من نفسه without resistance , من الطبيعي أنك ما تختبرهش against resistance

❖ Result of examination : Grades of muscle power:

Grade :	Muscle power:
0.	○ No muscle contraction visible (complete paralysis)
1.	○ Muscle contraction visible without joint movement (flicker; Twitches)
2.	○ Movement after elimination of effect of gravity
3.	○ Movement against gravity, but without resistance
4.	○ Movement against gravity, with some resistance
5.	○ Normal, Movement against gravity, with Full resistance

1. Upper limbs

1. Hand; fingers صوابك

Without resistance

Flexors (flexor digitorum)

أقلل - اتني - صوابك

With resistance

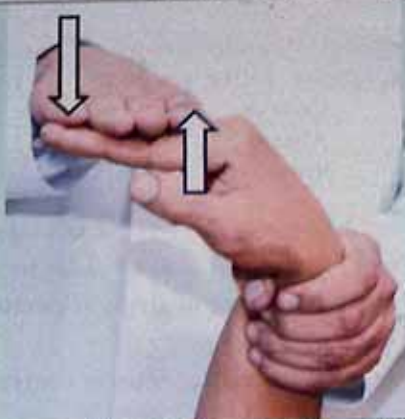


أقلل صوابك جامد على صوابي ..
متخلنيش أطلعهم قاومني (hand grip)
للتميز أوي كمل وقوله افتحهم بسرعه: ليه ؟
To exclude voluntary myotonia

Extensors (extensor digitorum) افتح

افرد - صوابك

resistance تعمل وإيدك الشمال تثبت ال wrist



افرد صوابك جامد ... متخلنيش اتنيهم ...
قاومني

Abductors (dorsal interossei)

أبعدهم - إيدك اليمين



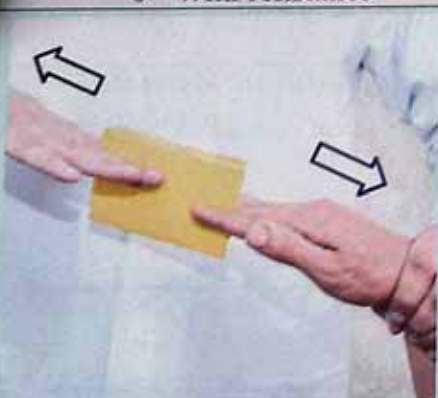
أبعد صوابك جامد ... متخلنيش اضمهم ...
قاومني

Without resistance

Adductors (palmar interossei)

ضمهم

With resistance



ضم صوابك على الورقة جامد ... متخلنيش
اشدها ... قاومني

Opposition (opponens pollicis)

سيح - عد على صوابك

resistance تعمل وإيدك الشمال تثبت ال wrist



اعمل حلقة بصباك الصغير على الكبير
ومتخلنيش أعدي منهم ... قاومني

Lumbricals : عمل وضع الكتابة



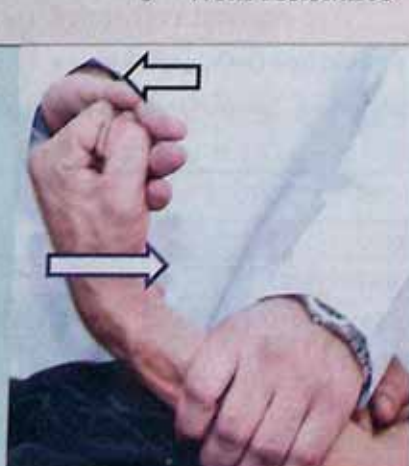
اعمل وضع الكتابة ... متخلنيش أعدي
صباكي من صوابك ... قاومني

2. Wrist : كفك (يفضل الإختبار والأصابع مقفولة)

Without resistance

Flexors (flexor carpi-radialis & ulnaris) اتني كفك

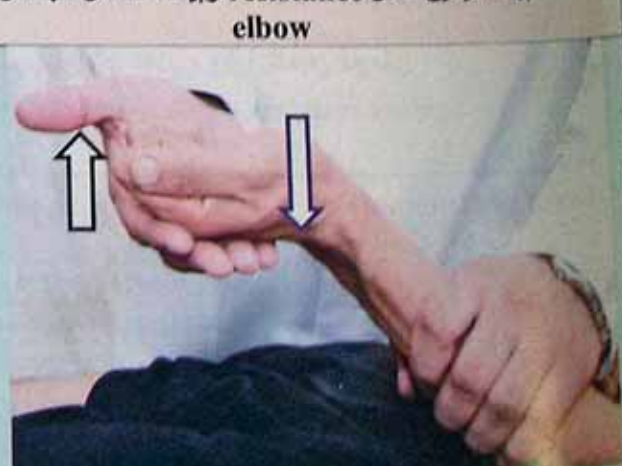
With resistance



اتني كفك جامد .. زق على إيدي

Extensors (extensor carpi-radialis & ulnaris) افرد كفك

إيدك اليمين تعمل resistance وإيدك الشمال تثبت ال elbow



افرد كفك جامد ... زق على إيدي ...

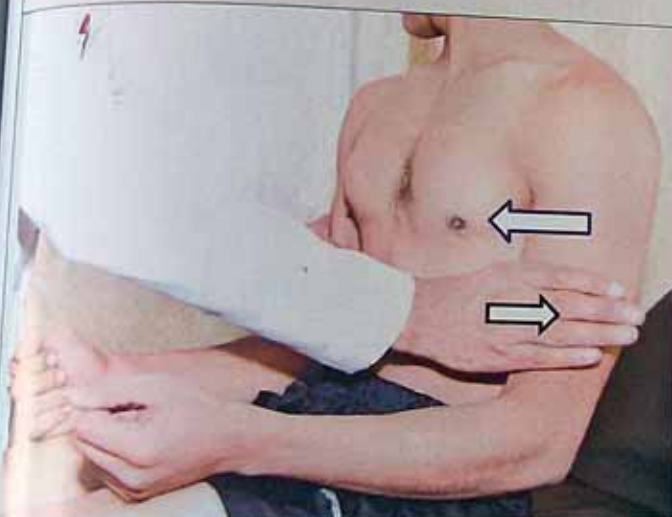
○ Flexors (biceps, brachioradialis) ○ Elbow: كوعك
 ⚡ With resistance ⚡ Without resistance



○ انتي كوعك جامد... شد على ايدي

3. Shoulder ○ افرد كوعك جامد... زق على ايدي
 (يفضل الفحص والمريض جالس ← لازم تقولها للدكتور): ذراعك

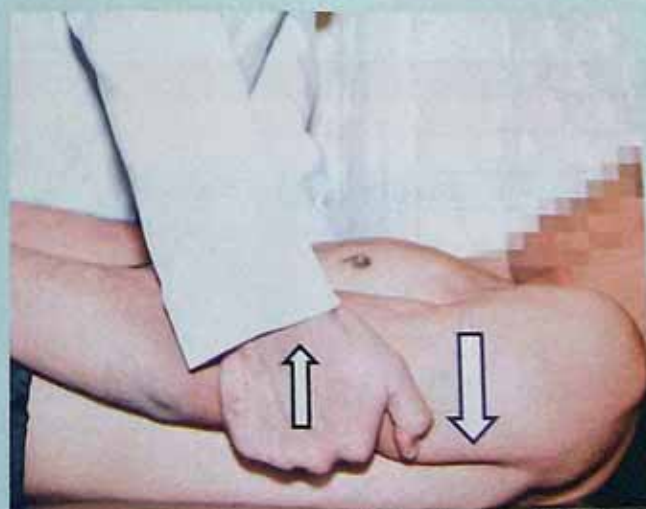
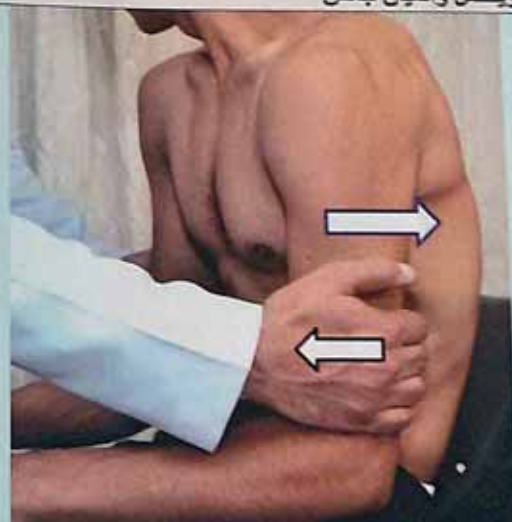
○ Flexors (anterior fibers of deltoid)
 ⚡ With resistance



⚡ Without resistance

○ Extensors (Posterior Fibers of deltoid)
 ذراعك لورا لو جالس أو تحت لو نايم

يتم فحص الذراعين معاً لأن مافيش مفصل تثبته
 اعمل إيدك زي الكماشة حول ال arm علشان تعمل resistance وتقول
 للدكتور ويفضل والعيان جالس



○ زق ذراعك لقدام لو جالس (أو لفوق لو نايم) جامد... متخلنيش انتز
 ... قاومني

○ زق ذراعك لورا لو جالس (أو تحت لو نايم) جامد... متخلنيش أرفعهم
 ... قاومني

Without resistance

○ Abductors (supraspinatus, deltoid, trapezius) جَنَح

○ Adductors (pectoralis major, pectoralis minor assisted by latissimus dorsi & teres major) ضم

يتم فحص الذراعين معاً لأن مفاصل مفصل تثبته
أعمل إيدك زي الكماشة حول ال arm علشان تعمل resistance من
غير ما تغير الوضع وتقول للدكتور ويفضل والعيان جالس

With resistance



○ جَنَح ذراعك لبره جامد ... زق إيدي لبره



○ ضم ذراعك لجوه جامد ... زق إيدي لجوه

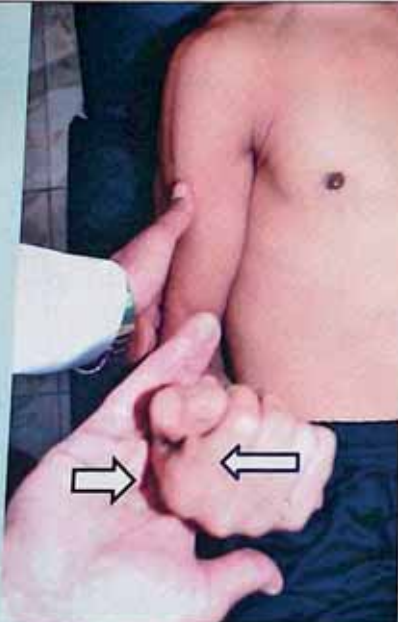
Without resistance

○ External rotation: سرح شعرك

○ Internal rotation:

خط إيدك في جيبك أو بإيدك اليمين المس جيبك الشمال والعكس صحيح.

With resistance



○ ضع ال arm وال forearm في زاوية قائمة: زق إيدي اليمين لبره جامد



○ ضع ال arm وال forearm في زاوية قائمة: زق إيدي اليمين لجوه جامد

II. Lower limbs

1. Toes: صواب رجلك

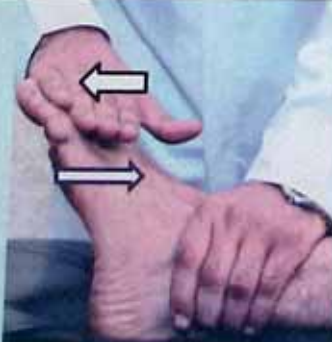
Without resistance

○ Dorsiflexion صواب رجلك لفوق

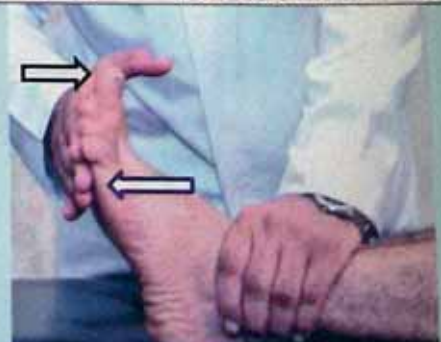
○ Planter flexion صواب رجلك لتحت

With resistance

إيدك اليمين تعمل resistance وإيدك الشمال تثبت ال ankle



○ ارفع صواب رجلك لفوق جامد ... شد على إيدي



○ نزل صواب رجلك لتحت جامد ... زق على إيدي

2. Ankle : وشن رجليك

Without resistance

Dorsiflexion (anterior tibial muscles : tibialis anterior, peroneus longus, brevis) وشن رجليك لافوق

With resistance



ارفع وشن رجليك لافوق جامد ... شد على ايدي

Plantar flexion (calf muscles; gastrocnemius, soleus, plantaris) وشن رجليك لتحت

ايدك اليمين تعمل resistance وايدك الشمال تثبت ال knee



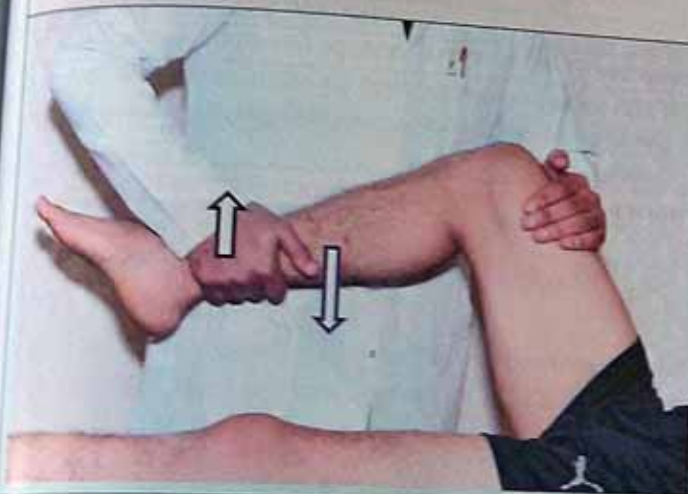
نزل وشن رجليك لتحت جامد ... زق على ايدي

3. Knee ركبتيك

Without resistance

Flexors (hamstrings: semi-tendonosus, membranousus, biceps femoris) انتي ركبتيك عليك

With resistance



انتي ركبتيك عليك جامد ... شد على ايدي

Extensors (quadriceps; vastus medialis, lateralis, intermedius, rectus femoris) افرد ركبتيك

خلي رجل العيان mid-position ايدك اليمين كماشة حول ال leg تعمل resistance وايدك الشمال تثبت ال hip



افرد ركبتيك جامد ... زق على ايدي

4. Hip رجليك

Without resistance

Flexors (ileo-psoas) ارفع رجليك

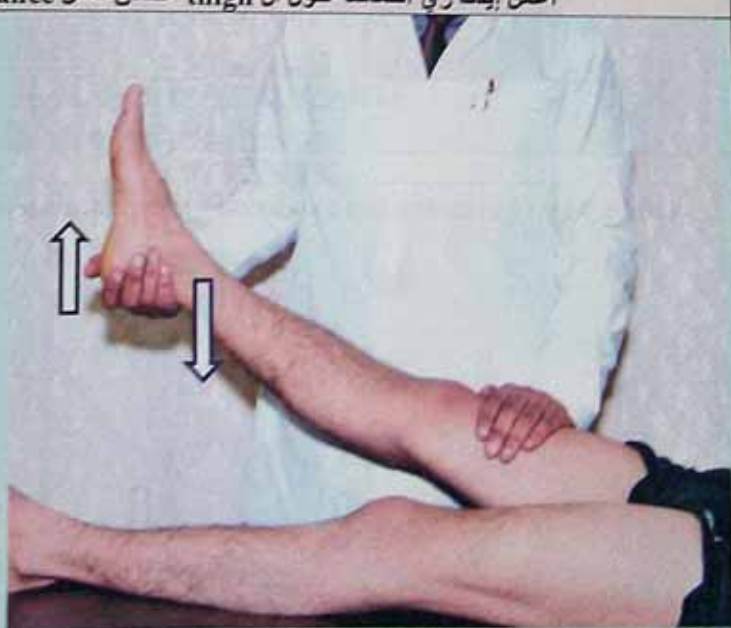
With resistance



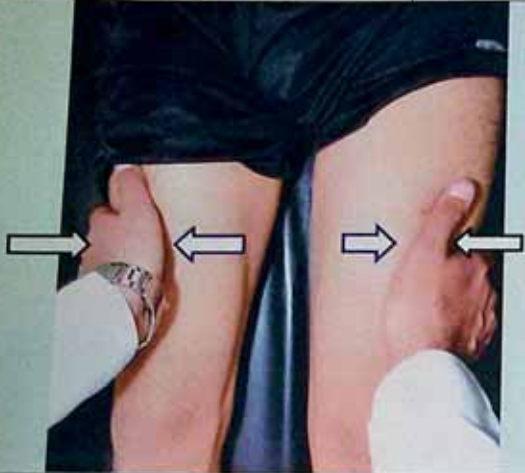
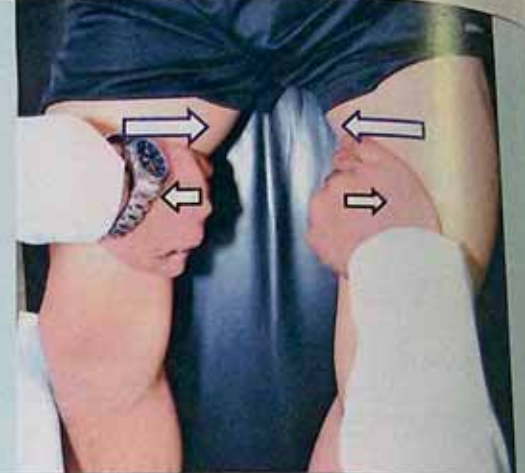
ارفع رجليك لافوق جامد ... زق على ايدي لافوق

Extensors (gluteus maximus) نزل رجليك

يتم فحص الرجلين معا لان مافيش مفصل تثبته اعمل ايدك زي الكماشة حول ال thigh تعمل resistance



نزل رجليك لتحت جامد ... زق على ايدي لتحت لها طريقة أخرى منتشرح بعد قليل والعيان نايم Prone

Without resistance ○ Abductors (gluteus medius, minimus) ابعاد - افتح رجلك	Without resistance ○ Adductors (adductor group; adductor magnus, longus, brevis, pectineus, gracilis) ضم رجلك يتم فحص الرجلين معاً لأن مفاصل تثبتته اعمل إيدك زي الكماشة حول ال thigh resistance تعمل
With resistance 	With resistance 
○ افتح رجلك جامد ... زق ايدي لبره	○ ضم رجلك جامد ... زق ايدي لجوه
Without resistance ○ External rotation: حط رجل على رجل	Without resistance ○ Internal rotation: والعيان قاعد على الكرسي قوله المس بركبتك اليمين الركبة الشمال والعكس
With resistance 	With resistance 
○ ضع ال thigh وال leg في زاوية قائمة: زق ايدي اليمين لجوه جامد	○ ضع ال thigh وال leg في زاوية قائمة: زق ايدي اليمين لبره جامد

❖ Other way to examine hip extension = examination of power in gluteus maximus :



- العيان ينام على وشه ... ليه ??? ← against gravity
- الدكتور يضع ايده اسفل العمود الفقري ويدوس ليه ???
To avoid action of trunk muscles
- خلي العيان يتني ركبتة ... ليه ???
To put the hamstring muscles out of work
- اقول للعيان ارفع رجلك لفوق ومتخلنيش أنزلها ... قاومني

III. Abdominal muscles : Without resistance

Flexors

العيان يرفع يديه أو يخطها تحت راسه وقوله هم يراسك



Extensors

العيان يخط يديه وراء راسه وقوله قوم طياره



With resistance

خط إيدك على جبهته أو راسه من واز وقوله هم ←
بص على ال Umbilicus :

❖ الإحتمالات:

- (1) العضلات التي فوق وتحت شفتين كويس ، أو مش شفتين كويس : ← umbilicus is central
- (2) العضلات التي تحت هي بين التي شفتة والتي فوق مش شفتة ← umbilicus is shifted downwards
- (3) العضلات التي فوق هي بين التي شفتة والتي تحت مش شفتة ← umbilicus is shifted upwards (Beevor's sign)

❖ Beevor's sign:

- **Definition :** spared upper fibers of rectus abdominis while lower fibers are paralyzed
- **Causes:** some cases of myopathy (pelvic girdle type)
- **Significance :** leveling sign: paraplegia at the level of T10

❖ Value of power examination :

1) Power grading

2) Distribution e.g. pyramidal tract lesion:

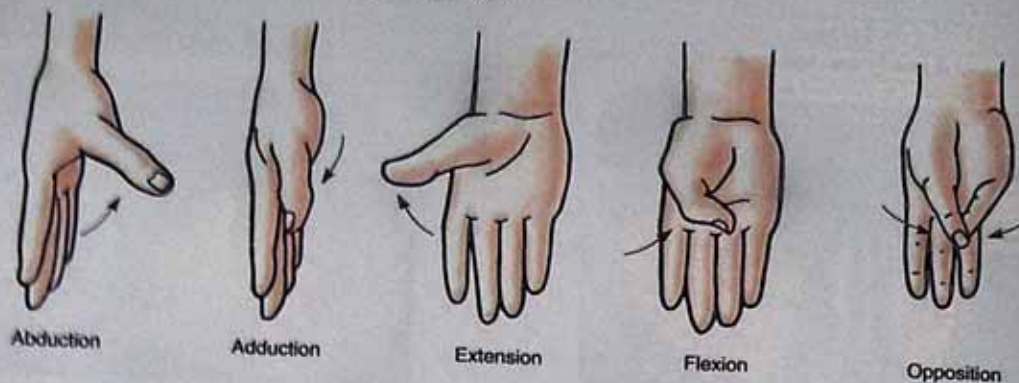
❖ In hemiplegia:

- ❖ Distal weaker than proximal & Abductors weaker than adductors in UL & LL.
- ❖ Extensors weaker than flexors in UL & Flexors weaker than extensors in LL

❖ S.B.: Types of muscles :

A. Antigravity muscles (Pull UP)	B. Progravity muscles (Pull Down)
Upper limbs	
Flexors, adductors	Extensors, abductors
Lower limbs	
Extensors, adductors	Flexors, abductors
Trunk	
Extensors	Flexors

- سؤال مستقل لوحدة في لجنة العملي
1. Without resistance : إدي العيان الأمر وأنت بتمثل الحركة قدامه :



2. With resistance :

• إيديك اليمين تعمل resistance وإيديك الشمال تثبت ال wrist

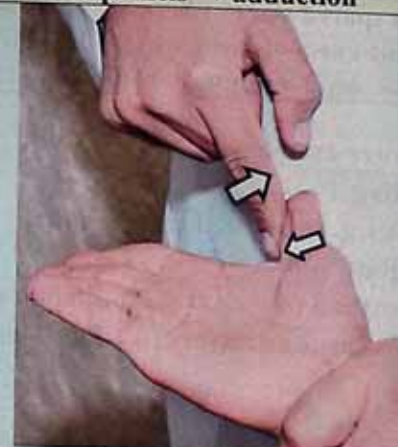
Muscles & action:

1) Opponens pollicis → opposition (see before)

2) Abductor pollicis longus → abduction

• عمل حلقة بصباغك الصغير على الكبير ومتخلنيش أعدي منهم ... قاومني

3) Adductor pollicis → adduction



• المقاومة تكون على جانبي ال thumb
• ارفع صباغك لفوق جامد ... ومتخلنيش أنزله ... قاومني

• المقاومة تكون على جانبي ال thumb
• ضم صباغك لجوه جامد ... ومتخلنيش ابده ... قاومني

4) Flexor pollicis longus → flexion

5) Extensor pollicis brevis → extension



• المقاومة تكون من الأمام
• اتني صباغك جامد ... متخلنيش افرده ... قاومني

• المقاومة تكون من الخلف
• افرد صباغك جامد ... متخلنيش اتنيه ... قاومني

❖ N.B.: abduction movement :

▪ From 0-15°	▪ Supra-spinatus
▪ 15-90°	▪ Deltoid
▪ 90-180°	▪ Trapezius

1. Co-ordination:

❖ لازم تختبرها بعد ال power ... ليه ؟
لانها يمنع الحكم عليها تماماً في وجود ال motor weakness

- ❖ Test for coordination **not done** in limb showing **paralysis**:
 - Deep sensation → مسئول عنه وهو مغمض
 - Cerebellum → مسئول عنه في كل الظروف

○ Hemiplegia : examined in healthy side only

○ Paraplegia : upper limbs only

❖ Every test should be repeated **2 times**

اعملهم مرة وهو مفتوح ومرة وهو مغمض

❖ Technique : اتكلم له وشاور فهمه :

○ Eye open to examine the cerebellum

○ Eye closed to examine deep sensation

A. Finger - to nose test:

وانت مفتوح هات صباعك من بعيد على مناخيرك (يعاد وهو مغمض)



B. Finger - to finger test:

وانت مفتوح هات صباعينك الإثنين من بعيد على بعض (يعاد وهو مغمض)



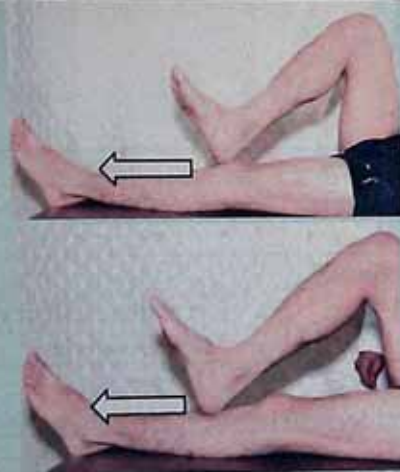
C. Finger - to fixed doctor's finger test:

وانت مفتوح هات صباعك من بعيد على صباعي



D. In LL: Heel - to knee test:

وانت مفتوح هات كعبك على ركبتيك ومشيه على قصبة رجلك (يعاد وهو مغمض)



❖ Result of examination; incoordination:

1. In UL:

- Intention Kinetic tremors which become more evident as the patient's forefinger approaches the target
- Decomposition of movement
- Dysmetria in the form of hypermetria or hypometria

2. In LL:

- As above + side to side movement

❖ Interpretation of results :

	Normal	Pure sensory ataxia	Pure cerebellar ataxia	Mixed ataxia e.g. Friedreich's ataxia
Eye opened	-ve	-ve	+ve	+ve
Eye closed	-ve	+ve	+ve (the same)	More +ve

❖ Confirmatory test for each type of ataxia :

I. **Cerebellar ataxia:** Signs of incoordination : when eye both opened & closed:

1) Eye : nystagmus, character of cerebellar nystagmus

○ On fixation	○ Horizontal	○ Biphase (jerky)	○ Rapid phase towards the fixation point	○ Bilateral
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2) Tongue: staccato speech

3) Neck: nodding

4) Trunk: titubation

5) Buttoning and unbuttoning test: failure is the earliest sign

6) Rebound phenomenon : Holme's sign :



• وش العين الناحية الثانية ... إخلع الساعة من إيدك ... لو العين لايس نضارة يقطعها ... ضع إيدك أمام وجه العين ... إحص العين :

• With sudden release of flexed elbow, the forearm may hit patient face

7) Dysdiadokokinesia : inability to perform rapid alternating movement e.g. pronation & supination

8) Gait: Walking along straight line, " heel to toe gait" "Tandem Gait" :

○ إشمعني المشية ديه ؟ لأنها تحتاج very high co-ordination



Cerebellar lesion	Disease	Gait disturbance
A. Archicerebellum:	Friedrich's ataxia	○ Standing : swaying (trunkal ataxia) ○ Walking : Wide base "drunken" or staggering gait
B. Neocerebellum:		
○ Unilateral	○ Cerebellar astrocytoma	○ Deviation to one side
○ Bilateral	○ Marie's ataxia	○ Zigzag gait

9) In both archicerebellar & neocerebellar lesions leads to :

• Hypotonia & hyporeflexia

• Pendulous Knee jerk

II. **Sensory ataxia:** Signs of incoordination :when eye closed only

1) Washbasin sign : is a fixed complaint of all patient , leading to swinging on washing face.

2) Rombergism : +ve Romberg's sign (see later)

3) Deep sensory loss: loss of vibration sense, position of movement, muscle sense & nerve sense.

4) Reflex: Hypotonia & hyporeflexia

5) Stamping gait: heavy strike of ground on walking due to lost deep sensation of ground

❖ Types of reflexes :

1. Reflexes

1. Deep 2. Pathological 3. Superficial

❖ قواعد فحص ال reflex :

1. لازم تعري العضلة وعينك عليها
2. على العضلة slightly stretched
3. بتعنى ال reflexes تحتاج stretch أكثر وده عن طريق الضغط بصياك على ال tendon ناحية اليمين بال index وناحية الشمال بال thumb
4. لازم التيان ميكونش tension ☞ قوله سيب نفسك شالص
5. دائماً قارن بين الناحيتين بنظام وأبدأ من الناحية السليمة
6. في حالات ال hyper-reflexia فقط ← إيض عن ال Clonus & Pathological reflexes
7. ال Clonus & Pathological reflexes تتصل في الناحية ال diseased only ومتصلش في الناحية ال normal
8. كل ال pathological reflexes بنستخدم فيها صباعا ماعدا ال supra-spinatus
9. لو الدكتور قالك اصل ال reflex الفلاقي لازم تقول ال center
10. أثناء الفحص لو reflex ما يظهرش اصل Reinforcement : Jandrassik's maneuver

→ Reinforcement : Jandrassik's maneuver:

- A. If the reflex is in the UL ☞ جز على سنالك جامد
- B. If the reflex is in LL ☞ جز على سنالك جامد وشبك وشد إيدك كده
- Mechanism of reinforcement: ↑ discharge in gamma fibers which supply reflex
- Result :
- A. If still negative after reinforcement ☞ areflexia
- B. If become positive after reinforcement ☞ hyporeflexia i.e. hyporeflexia is a reflex only obtainable after reinforcement

❖ Center of reflexes (root value):

❖ Center:	❖ Reflex:
C3-4	Supraspinatus
C5	Deltoid
C5 – T1	Pectoral
C5-6	Biceps – Brachioradialis
C6-7	Triceps
C8 – T1	Wrist clonus Finger flexion Hoffman Wetenberg
T6-T12 Upper : T6-T8 Middle : T8-T10 Lower: T10-T12	Abdominal
L1	Cremastric
L2-3-4	Tendon Knee jerk, Patellar, patellar clonus
L4	Adductor
L4-5	Gluteal
S1, S2 mainly S1	Ankle, Ankle clonus & Planter (Babinski)
S3,4,5	Anal

❖ Grades of deep reflexes:

Grade of :	Reflexes
0	Areflexia
1	Hyporeflexia (0/1)
2	Normal
3	Hyperreflexia
4	Clonus

❖ Technique : see below

I. Upper limbs :

Normal Deep reflexes :

1) Brachioradialis reflex or Supinator reflex: (C5-6)



What is inverted supinator reflex?

- Causes : lesion at C5 segment & root
- Components :
 1. Lost biceps reflex
 2. Exaggerated triceps reflex
 3. Inverted supinator reflex (flexion of fingers instead of flexion of elbow)

- الكوع في زاوية 120°
- اليد العيان mid-way between supination & pronation
- اخبط بالناحية العريضة على
- 1 inch above radial styloid process

2) Biceps reflex (C5-6)



3) Triceps reflex (triceps brachialis reflex) (C6-7)



- الكوع في زاوية 120°
- زود ال stretch بصباك
- اخبط بالناحية المديبة وعينك على ال biceps

- الكوع في زاوية 90°
- ممكن تعمل more stretch بأنك تشد على إيد العيان
- اخبط بالناحية العريضة وعينك على ال triceps

Pathological reflexes normally absent, if present they denote UMNL, done only in hyperreflexia:

1) Supraspinatus (C3-4)



- العيان قاعد والكتف مكشوف
- حس ال spine of scapula واخبط فوقه

2) Finger flexion (C8 - T1)



- اثني اصابع اليد بزاوية 90° وحط صوابك ضد صوابه كاتك هتبوس ايده
- اخبط على صوابك من تحت
- لو +ve هيشد على صوابك أو يقلل صوابه

3) Wetenberg (C8 - T1)



- شد صوابك من صواب العيان بتاعة نفس اليد (اليمين باليمين والشمال بالشمال)
- انتة شد وهو يشد
- لو طبيعي ← extension of the thumb, abduction
- لو hyper-reflexia ← flexion of thumb, adduction

4) Hoffman's (C8 - T1)



- امسك الصباع الأوسط بتاع العيان وثبت ال PIPJ واثني ال DIPJ وسيبها فجأة كاتك بتطرقها
- لو +ve صواب العيان تتقلل وخصوصا يحصل Flexion of thumb

5) Pectoral (C5-T1)



- حط صباك على ال anterior axillary fold واخبط فوقه

6) Deltoid (C5)



- حط صباك على ال deltoid واخبط فوقه

Search for clonus only in hyperreflexia : WRIST CLONUS, Organic sustained clonus is the surest sign of UMNL : see later.

II. Lower Limbs:

Normal Deep reflexes :

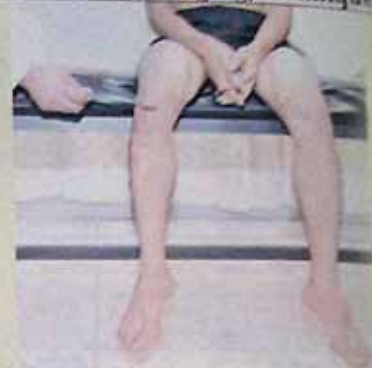
1. Tendon Knee jerk (quadriceps reflex) (L2,3,4)

A. Ordinary method



- اتني الرجل 120° على الناحيتين معاً
- ارفع الرجلين مع بعض من تحت الركبة بحيث تجعل كعبه يا دوب ملامس السرير
- حط ال thumb بتاع الايد اللي انت رافع بيها بين ال knees 2
- ابحث عن ال tendon بايدك الثانية وهو الواقع بين بروتين واحد أعلى الركبة والآخر أسفلها (هامة)
- اخبط بالناحية العريضة على ال tendon وعينك على ال quadriceps

B. Dangling (hanging) technique :



- الرجل متكلية عاملة زاوية 90°
- ابحث عن ال tendon بايدك وهو الواقع بين بروتين واحد أعلى الركبة والآخر أسفلها (هامة)
- اخبط بالناحية العريضة على ال tendon وعينك على ال quadriceps

2. Ankle reflex (triceps surae reflex, gastrocnemius soleus reflex) (S1, 2 mainly S1)

A. Ordinary method



- اتني الرجل 90° ليه؟ ابطل action of gastrocnemius on knee وخليه كأنه حاطط رجل على رجل وينيمها لبره
- اعمل dorsiflexion علشان تعمل slight stretch
- اخبط بالناحية العريضة على tendo-achillis وعينك على ال calf muscles
- لو الدكتور سالك هتشوف ايه : قوله plantar flexion & eversion

B. Kneeling technique :



- العيان قاعد على حاجة مستواها عالي بحيث رجليه متكونش لامسة الارض مع نفس الخطوات السابقة

Pathological reflexes normally absent , if present they denote UMNL, done only in hyperreflexia:

1. Adductor (L4)



- اعمل abduction و external rotation لل hip
- دور على ال tendon وده انك تطلع سنة فوق ال adductor tubercle
- واعمل rolling هاتحس كأن في حبل تحت ايدك
- دوس عليه بصباك جامد علشان تزود ال stretch ثم اخبط
- لو hyper-reflexia ← adduction of thigh

2. Patellar (quadriceps reflex) (L2,3,4)



- ركية العيان مفرودة 180°
- حط your index بسميفه above patella واخلط عليه من أعلى لأسفل
- لو hyper-reflexia ← الركبة تنط لفوق

Clonus in Limbs:

Search for clonus only in hyperreflexia : Organic sustained clonus is the surest sign of UMNL.

1. Wrist clonus (C8-T1)



اسند ال forearm بإيدك الشمال من الكوع وامسك ايده باليمين وطلعها لقدام حمله بسيطة وبعدين ارجع اعمل dorsiflexion فجأة

Types of clonus :

- 1) Sustained , non-sustained على طول , مرة أو مرتين
- 2) Organic , hysterical عضوي , هستيري

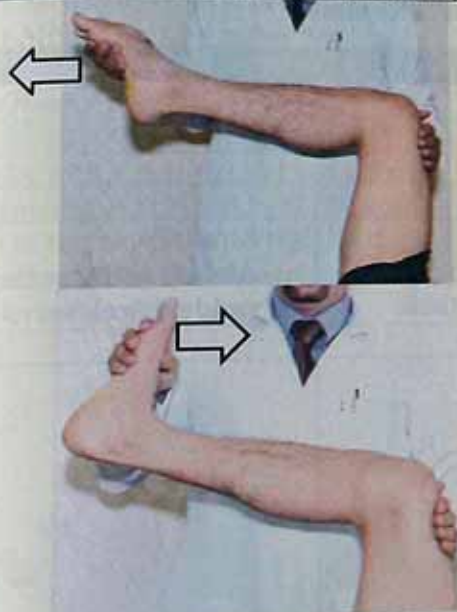
→ DD between organic & hysterical clonus :

- تلقى ال stretch التي أنته عامله بدون ما يشعر المريض
- لازم العيان يحس أنك لسه حاطط إيدك وفي نفس الوقت شد الناحية العكسية
- hysterical لو استمر : يبقى hysterical
- لو اختفى يبقى organic

• Organic sustained clonus is the surest sign of UMNL

• كل clonus معاه UMNL وليس العكس

2. Ankle clonus (S1, 2 mainly S1)



- اتني رجله لقدام حمله بسيطة وبعدين اعمل dorsiflexion فجأة
- تلاقي القدم بترقص فوق وتحت

3. Patellar clonus (L2,3,4)



- وشك لرجل العيان
- خلي صوابك مع بعض في مواجهة ال thumb زي الكماشة
- شد جلد الركبة لفوق شوية
- ثم زق الركبة لتحت هتلاقي الركبة بترقص طالعة نازلة

N.B.: Other Pathological reflexes (normally absent) : in the face

1. Pouting or snout

- Light tapping of the closed lips near the midline by finger or by handle of the hammer → upward movement of lips



2. Palmo-mental

- Scratch thenar eminence → contraction of the mentalis muscle of the chin



❖ Superficial reflexes: Planter, Abdominal, Cremasteric, gluteal & Anal.

Superficial reflex : Planter reflex (S1, 2 mainly S1)

Normal: Flexor response



Abnormal: Extensor response



فكرته :
بطريقة ما
stimulate
قدم
العين وشوف
التنحية في الصباع
الكبير

I. Technique of Babinski method:

أخذي رجل العين مفرودة على السرير واستخدم مفتاح وامشي على ال lateral side من القدم ثم لف مع نهاية الصواب وقف قبل بداية الصباع الكبير

1) Normal: Flexor response : planter flexion of toes

2) Abnormal :

A. Extensor response = Babinski sign = +ve Babinski
: dorsiflexion of big toe ($\pm$ fanning of other toes):

❖ Causes :

1. Δ ($\pm$ extra Δ) tract lesion
2. Infants : 1st year of life
3. Under anesthesia
4. Deep sleep & Deep coma

→ It is considered sure sign of UMNL

❖ N.B.: -ve Babinski اسمها مافيش حاجة اسمها

B. Absent محصلش حاجة :

❖ Causes of absent (Equivocal) response:

- Loss of sensation over the sole of the foot
- Cold or thick skin
- LMNL at S1
- Shock stage of UMNL
- Total paralysis of extensors of big toe e.g. myopathy
- Marked foot deformities

II. Other Techniques to elicit planter reflex:

A. Indications:

- 1) Babinski can't be done e.g. injury to the sole of the foot
- 2) For reinforcement & Equivocal Babinski

B. Techniques :

1) Shaddock:

- Lateral aspect of dorsum of foot from lateral malleolus to little toe

2) Barada:

- Lateral border of the foot

3) Gonda:

- 3rd & 4th toes are passively flexed then suddenly release

4) Stransky:

- Abduct little toe then suddenly release

- Red line : Shaddock
- Green Line : Barada



5) Oppenheim:

- Firm pressure in the lower part of the shaft of the tibia



6) Gordon:

- Calf muscles are firmly pinched



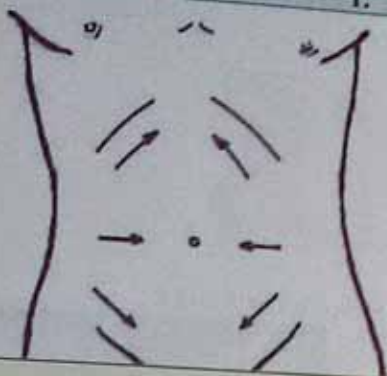
7) Schaeffer:

- Tendon Achilles is firmly pinched



Superficial reflexes :

1. Abdominal



❖ Technique :

- (1) اكشف بطن العيان ← relax abdominal muscles حاول انك
- (2) استخدم دبوس أو نهاية tongue depressor مكسورة
- (3) اسعمل scratch من برة لجوه في ثلاثة level على الناحيتين

❖ Center : T6-T12

- Upper abdomen : T6-T8
- Middle abdomen : T8-T10
- Lower abdomen: T10-12

❖ Normal response: contraction of abdominal muscles ± pull of the umbilicus towards the stimulator

❖ Significance : leveling in paraplegia

❖ Lost abdominal reflexes :

1. UMN above level of segmental supply of reflex
2. LMNL affecting reflex arc itself
3. Severe weakness & wasting of muscles e.g. myopathy
4. Obesity & ascites

❖ Exaggerated abdominal reflexes :

- Psychoneurosis

2. Cremasteric (L1)

- Stroke with a pointed but not sharp instrument over the skin of the upper part of the medial aspect of the thigh → contraction of cremasteric muscles → elevation of the testicle

3. Gluteal (L4,5)

- Stroke with a pointed but not sharp instrument across one buttock → contraction of the ipsilateral gluteal muscle

4. Anal (S3,4,5)

- Scratch the skin of the perianal region → contraction of external anal sphincter

DISCUSSION ON MOTOR EXAMINATION & REFLEXES

❖ What are the causes of muscle wasting ?

- 1) Bilateral symmetrical, proximal : myopathy
- 2) Bilateral, symmetrical, distal : Peripheral neuropathy
- 3) Diffuse : UMNL
- 4) Patchy or segmental : AHCs lesions
- 5) Generalized : elderly, malnutrition, malignancy



❖ What are the causes of wasting of small muscles of hand? Supplied by C8-T1

- 1) AHCs lesions e.g. poliomyelitis & syringomyelia
- 2) Anterior roots & spinal nerve lesion e.g. cervical spondylosis
- 3) Lower brachial plexus lesion: cervical rib
- 4) Peripheral neuropathy & carpal tunnel syndrome
- 5) Myasthenia gravis
- 6) Muscle lesion: distal type of myopathy "Gower disease"
- 7) Others : rheumatoid arthritis & myxedema



What are the causes of Pseudohypertrophy?

- 1) Segmental : Duchenne & Becker myopathy
- 2) Generalized : myotonia congenita "young Herculean", myxedema & acromegaly.



❖ What are the causes of pes cavus?

- 1) Fredrieich's ataxia
- 2) Peroneal muscle atrophy
- 3) Duchenne myopathy
- 4) Syringomyelia
- 5) Severe wasting of intrinsic foot muscles



❖ What are the diseases associated with marked tropic changes?

○ Cauda equina	○ Syringomyelia	○ Tabes dorsalis
○ Peripheral neuritis especially diabetic, leprotic		

❖ What do you know about Gower ?

1. Gower sign : climbing test
2. Gower test : muscle tone at shoulder
3. Gower disease : distal type of myopathy

❖ What is meant by tonic atrophy & causes?

- Tonic atrophy : combined signs of LMNL in the form of wasting of muscles with signs of UMNL in the form of hypertonia and hyperreflexia
- Causes:

1. Posterolateral cervical spondylosis	2. Amyotrophic lateral sclerosis
--	----------------------------------

❖ What are the sure signs of pyramidal lesion (UMNL)?

1. Plantar reflex: Babinski sign
2. Organic sustained clonus
3. In bilateral pyramidal lesion : precipitancy , positive jaw jerk

❖ What are the causes of ?

Hypotonia	Hyporeflexia
1. LMNL : lesion of the reflex arc:	
A. Afferent (sensory) limb : PN & tabes dorsalis	
B. Center (AHC) : poliomyelitis & MND	
C. Efferent (motor) limb: PN	
D. Effector (muscle): myopathy & myasthenia gravis	
2. Posterior column lesions	
3. Cerebellar lesions (ataxia)	
4. Extrapyramidal lesions (chorea)	
5. Shock stage of UMNL	
	Extrapyramidal lesion: parkinsonism

❖ What are the causes of ?

Hypertonia	Hyperreflexia
○ Pyramidal lesion, i.e. UMNL (spasticity)	
○ Extrapyramidal lesion (rigidity) i.e. parkinsonism	○ Tetany & Thyrotoxicosis
○ Meningeal irritation, Myotonia, Catatonia & Hysterical	

❖ What are the differences between spasticity & rigidity ?

	Spasticity	Rigidity
▪ Cause	○ Pyramidal lesion	○ Extrapyramidal lesion
▪ Distribution	○ Antigravity muscle: - Flexors of UL - Extensors of LL & trunk ○ Distal > proximal	○ Antigravity & progravity muscle: - Flexors of UL, LL & trunk ○ Proximal > distal
▪ Character	○ Clasp – knife	○ Lead pipe: uniform resistance ○ Cog wheel: resistance is intermittent & interrupted by tremors
▪ Deep reflexes	○ Hyperreflexia	○ Hyporeflexia

❖ Cause of hypertonia and hyporeflexia? Parkinsonism

❖ What are the causes of ...?

❖ Lost ankle preserved knee reflex	❖ Lost ankle exaggerated knee reflex	❖ Lost knee & preserved ankle reflex	❖ Lost knee exaggerated ankle reflex
• Friedreich's ataxia	• Friedreich's ataxia	• Cauda equina (L2,3,4)	• Paraplegia + cauda affecting L2,3,4
• SCD	• SCD	❖ What are the causes of pendular knee jerk ?	
• Peripheral neuropathy	• Pellagra		
• Epiconus lesion			
• Cauda equina lesion of S1			
• Holmes - Adies myotonic pupil		• Cerebellar ataxia	
		• Chorea	

2. Examination of sensory system

1) Superficial sensation 2) Deep sensation 3) Cortical sensation

1) Superficial sensation:

1) Pain	2) Touch	3) Temperature
استخدم دبوس	استخدم فرشاة أو قطن	باستخدام أنابيب اختبار تحوي ماء بارد وماء ساخن

1. Compare both sides of body between right & left side; starting by healthy side :

At the level of :	Value :
Head , trunk (chest) , UL (arm) , LL (Leg)	Hemi-anesthesia (hemiplegia)

2. Compare between Head , trunk (chest, abdomen) , UL (arm) , LL (Leg) of same side and after finishing do the opposite side :

- Better UL (arm) & upper trunk (chest), less in lower trunk (abdomen) & LL (Leg) → extra-medullary lesion → **sensory level of paraplegia (detect the level)**
- Better in LL (Leg) & lower trunk (abdomen) less in upper trunk (chest) & UL (arm) & → intra-medullary lesion → **jacket sensory loss of paraplegia**

→ Technique of sensory level:

إبدأ من رجله صاعداً لأعلى وقوله هاشكك ولما الإحساس يزيد قول وشاور

↳ Radicular supply of the trunk:

Up to Axilla : T2	Up to Nipple: T5 (T4)
Up to Costal margin: T6	Up to Umbilicus: T10
Up to Inguinal ligament :T12	

3. Compare distal with proximal part of the same limb, after finishing do the opposite side:

- Value : detect the level of **stock & glove hyposthesia** in peripheral neuropathy

إبدأ من very distal واطلع حته حته

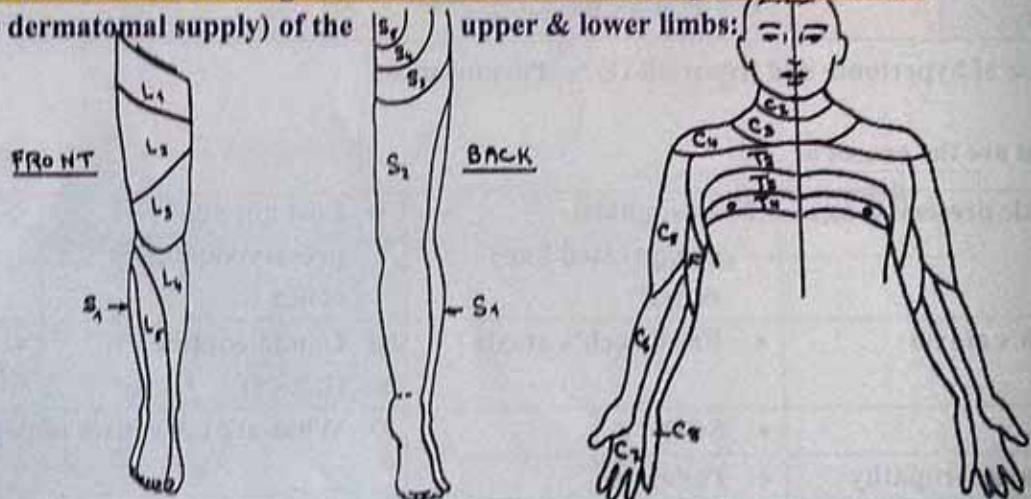
قول للعيان هاشكك ولما الإحساس يزيد قول وشاور , علشان تطلع ال level بتاع الجوانتي والشراب

4. Circumferential comparison between lateral, medial, posterior aspects of the limb, after finishing do the opposite side:

- If sensation is defective in one dermatome : radicular sensory loss in that dermatome as in cauda equina lesion

⚠ If the 4 above screening tests were normal → intact superficial sensation

↳ Radicular supply (= dermatomal supply) of the upper & lower limbs:



Upper limb		Lower limb	
C2	Angle of mandible	L1	Upper 1/3 front of thigh
C3	Neck	L2	middle 1/3 front of thigh
C4	Shoulder	L3	Lower 1/3 front of thigh
C5	Lateral aspect of arm	L4	Big toe
C6	Thumb	L5	Middle 3 toes
C7	Middle 3 fingers	S1	Little toe
C8	Little finger	S2	Posterior aspect of thigh, leg & sole of foot
T1	Medial aspect of arm	S3,4,5	Around anus
T2-7	Thorax	T8-12	abdomen

2) Deep sensation:

1) Sense of position	2) Sense of movement	3) Vibration sense (bone sense)	4) Muscle sense (pressure sense)	5) Nerve sense (electric sense)
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❖ N.B.:

- ❖ Joint sense = sense of position + sense of movement
- ❖ Deep pain sensation = muscle sense + nerve sense

1. Joint sense :



- ❖ فكرته: وانت مغمض عينك تحس مكان صوابك وأطرافك وهل هي متحركة ولا ثابتة
- ❖ طريقة الفحص:
 - 1) لو يتم الفحص في الـ LL استخدم الـ big toe , لو يتم الفحص في الـ UL استخدم الـ index finger
 - 2) امسك صباع رجله الكبير من الجانبين وبإيديك الثانية ضم الصواب الأخرى to avoid friction
 - 3) اعط العيان مثال وهو مفتاح عينية وقوله كده يعني فوق وكده يعني تحت , كده بيتحرك , كده واقف
 - 4) ثم قوله غمض عينيك وأسأله شويه

2. Vibration sense

❖ Using tuning fork 128 Hz/ sec

- ❖ Test : Place it over the following bony prominence :
- Medial malleolus: peripheral nerve
 - ASIS (anterior superior iliac spine): posterior column
 - Forehead: thalamus



- ❖ طريقة الفحص :
- 1) إبدأ بالـ forehead:
 - اخبط الشوكة وحطها على الـ forehead وأسأله حاسس بايه لازم يقولك حاسس بزنة
 - لو مقالش كذا قوله ديه زنة واخبطها مرة ثانية وخليه يحسها
 - لو حاسس تبقى الـ thalamus سليمة
- 2) ثم اختبر الـ medial malleolus:
 - لو حاسس يبقى الـ posterior column والـ peripheral nerve سليم
 - لو مش حاسس يبقى في الـ lesion في الـ posterior column أو الـ peripheral nerve
- 3) اختبر الـ ASIS:
 - لو مش حاسس يبقى الـ posterior column lesion
 - لو حاسس يبقى الـ peripheral neuropathy

تجرب هل ممكن تستخدم الـ vibration sense في الـ UL ؟ نعم :

- Radial styloid process & olecranon: peripheral nerve
- Clavicle: posterior column

3. Muscle sense :

❖ Technique :



- تجرب العيان بيتني رجله نصف تنية وتضغط على الـ calf muscle ضغطه بسيطة
- تجرب وبص على وش العيان وشوفه رد فعله ... طبيعي ولا هيتالم ولا مش هيتالم
- تجرب لو لقينه متالمش إسأله حاسس بضغط ايدي ولا لا علشان تتأكد

- Normally: disagreeable sense
- Exaggerated : tender calf muscles ; causes: DM, DVT, myositis, Landry - Guillan - Barre syndrome
- Lost muscle sense : Abadie's sign → neurosyphilis

4. Nerve sense : rolling of the ulnar nerve or lateral popliteal nerve against bone → electric sense

5. Romberg's test:

- Done when one or more of the deep sensations are lost



- ❖ فكرته:
 - الشخص الطبيعي لو قام ووقف وغمض عينه ← يحصل حاجة لأن الـ deep sensation عنده سليمة , بينما لو كانت الـ deep sensation عنده متأثرة العيان يفقد توازنه ويقع

❖ طريقة الفحص : اطلب من العيان:

- 1) يقف
- 2) يضم رجله
- 3) الدكتور يحوط العيان بإيده
- 4) العيان يغمض عينه

❖ Result : If the patient begins to shake or loses his balance → +ve Romberg's sign

3) Cortical sensation:

شروط الفحص :
 لازم يكون ال superficial & deep sensation سليم
 لازم العيان يقلل عينيه في كل الفحوصات
 تتعمل في الناحية ال normal ومتتملش في الناحية ال diseased

1. Tactile localization :point localization

شكه بالدبوس في ايده وقوله الشكه فين



2. Tactile discrimination (2 points discrimination):

شك العيان شكتين في نفس الوقت
 المسافة ما بين الشكتين 4 سم (ولو في الظهر 10 سم)
 اسأل العيان دول واحدة ولا اتنين



3. Stereognosis:

حظ حاجة معرفة في ايد العيان (قلم مثلا) واسأل ده ايه



4. Graphosthesia:

إرسم دايره (أو اكتب رقم) على كف العيان واسأله انا رسمت ايه ؟



5. Perceptual rivalry (sensory inattention = sensory extinction)

شك العيان شكتين بنفس القوة في نفس الوقت في نفس المكان على الناحيتين في كل من كفيه أو رجله
 واسأله حاسس بواحدة ولا اتنين
 لو حس بالأتنين يبقى ← cortex سليم
 لو حس بشكه واحدة يبقى ← cortex مضروب (cortical sensory loss)



DISCUSSION ON SENSORY EXAMINATION

❖ What are types of hyposthesia associated with UMNL & LMNL?

- ❖ Types of hyposthesia associating with UMNL:
- 1) hemihyposthesia : hemiplegia
 - 2) Sensory level : paraplegia
 - 3) Jacket of dissociated sensory loss : Syringomyelia Intramedullary lesions

❖ Types of hyposthesia associating with LMNL:

- 1) Glove & stock : PN
- 2) Radicular sensory loss : cauda equine & spondylosis
- 3) Saddle – shaped area : cauda equine & conus medullaris

❖ What are the causes of thickened nerve?

- 1) Interstitial polyneuropathy
- 4) Acromegaly

- 2) Peroneal Muscle Atrophy
- 5) Myxedema

- 3) Amyloidosis
- 6) Neurofibromatosis

❖ What are the patterns of sensory loss & the site of the lesion?



1. 2. 3.



4.



5.



6.



7.



8.



9.



10.

❖ Pattern of sensory loss

❖ Site of the lesion

- 1) Mononeural
- 2) Stock & glove
- 3) Maculo- anesthetic { Patchy loss in leprosy}

- Peripheral nerve lesion

- 4) Radicular loss

- Root lesion

- 5) Saddle loss

- cauda & conus

- 6) Hemi hyposthesia of dissociative nature (Brown - Sequard syndrome)

- Unilateral cord lesion

- 7) Sensory level

- Extra-medullary lesion

- 8) Jacket sensory loss

- Intra-medullary lesion

- 9) Crossed hemihyposthesia

- Lateral medullary syndrome

- 10) Hemihyposthesia

- Capsular & brain stem lesions

- 11) Cortical sensory loss

- Area 1, 2, 3 of parietal lobe

❖ Causes of hyposthesia of dissociated nature :

❖ Neurological diseases with intact sensation:

- Loss of pain & temp, with preserved deep sensation & fine touch in :

- 1) Syringomyelia & syringobulbia
- 2) Brown Sequard syndrome
- 3) Intramedullary tumors
- 4) Cerebellar artery occlusion syndrome

- 1) Muscle disease (myopathy, myotonia, myasthenia gravis & myositis)
- 2) Motor neuron disease
- 3) Marie's ataxia
- 4) Parkinsonism, chorea
- 5) Poliomyelitis
- 6) Hereditary spastic paraplegia

❖ Causes of motor neuropathy	❖ Causes of sensory neuropathy
• Acute infective polyneuritis • Lead neuropathy • Diphtheritic neuropathy • Porphyria	• Alcoholic neuropathy • Arsenic neuropathy • Leprotic neuropathy • Diabetic neuropathy • Vitamin deficiency neuropathy

3. Examination of Others : Cranium , back, spine & Gait

❖ Examination of cranium	❖ Examination of back & spine: العيان وبص على ظهره
1. Size 2. Shape, sutures, fontanel 3. Bony bosses e.g. meningioma 4. Tenderness, dilated veins, arterial bruit, nevi 5. McEwen sign in brain tumors: Tapping the skull → resonant sound	• In cases of paraplegia, cauda equina, cervical spondylosis: 1. Tenderness 2. Deformity 3. Hair tuft (spina bifida) 4. Swelling 5. Abnormal pigmentation "cafe' au lait" of Neurofibroma

❖ Examination of Gait : يقوم العيان ونبص على مشيته

Done if the patient can walk		
Lesion	Cause	Gait
1) UMN		
A. Unilateral	○ Hemiplegia	○ Circumduction
B. Bilateral	○ Paraplegia	○ Scissoring
2) LMN		
A. Muscle	○ Myopathy	○ Waddling
B. Peripheral Nerve	○ Peripheral neuropathy	○ High steppage
3) Posterior Column	○ SCD, Tabes dorsalis	○ Stamping = strike forcibly ground
4) Cerebellum		
A. Archicerebellum	○ Friedreich's ataxia	○ Standing : swaying (trunkal ataxia) ○ Walking : Wide base "drunken" or staggering gait
B. Neocerebellum		
○ Unilateral	○ Cerebellar astrocytoma	○ Deviation to one side
○ Bilateral	○ Marie's ataxia	○ Zigzag gait
5) Extra pyramidal	A. Parkinsonism	○ Short steppage (mild) ○ Shuffling (moderate) ○ Festinant (severe)
	B. Chorea	○ Dancing
6) Hysterical	❖ Psychological : bizarre, non-injurious & suggestible e.g. : ○ Pushing the paralyzed limb in front or dragging it behind ○ Astasia abasia : inability to stand or walk ○ Tandem walking : ask for heel to toe walk, This exaggerate instability	

4. Examination of Other Systems:

- Examine all systems as mentioned in general examination, but stress on the following :
 - In cases of paraplegia : examine chest (TB)
 - In cases of hemiplegia : examine heart (MS, hypertension)
 - In cases of DM examine all the body for complications of DM

Diagnosis Of Neurological Case

a. Etiological Diagnosis • Heredofamilial • Symptomatic (vascular, traumatic, inflammation)	b. Anatomical Diagnosis • Where is the lesion : - Cortical, spinal cord, cerebellum	c. Functional Diagnosis: • Stage : ○ Stage of neuronal shock ○ Stage of neuronal recovery ○ Stage of improvement with residual • Paralysis • Complication
C. Pathological Diagnosis ❖ What is the lesion: • Focal : affection of anatomically adjacent structures: - Usually asymmetrical, with sensory or motor level ▪ Systemic : affection of physiologically related systems: - Usually bilateral & symmetrical, without a level, Gradual onset & slowly progressive course ▪ Disseminated : affection of more than one focus which are neither anatomically nor physiologically related - Usually asymmetrical - Without a level - Rapid onset & intermittent course		

❖ Neurological cases:

- Hemiplegia, Brown Sequard syndrome, Paraplegia, Peripheral neuropathy, Peroneal muscle atrophy, Myopathy, Ataxia, Motor neuron disease, Parkinsonism, Disseminated sclerosis (multiple sclerosis)

❖ Summary of neurological examination:

I. General examination:

- | | | | |
|---------------|--------------------|---------------------|---|
| ○ Vital signs | ○ General overview | ○ Systemic overview | ○ Other systems except that of local exam |
|---------------|--------------------|---------------------|---|

II. Local examination : neurological examination

1. Mentality

- | | | | |
|--------------------------|---------------------------------------|--------------------------------------|-----------------|
| ○ State of consciousness | ○ Orientation to time, place & person | ○ Memory : immediate, recent, remote | ○ Affect (mood) |
| ○ Mentality | ○ Behavior (cooperative) | | |

2. Speech

- Aphasia, (Dysarthria : slurred, staccato; monotonous, scanning)

3. Cranial nerve examination

A. CN I: Smell

B. CN II: acuity of vision, field of vision (confrontation test)

C. Ocular nerves : 3,4,6 CN

- ❖ Inspection : ptosis, squint, size of pupil, nystagmus

	Each eye separately	Both eye together (conjugate eye movements)	
❖ Power	Accommodation (near)	Pupillary light	Ciliospinal
❖ Reflexes	D. CN 5	E. CN 7	F. CN 9,10
❖ Inspection	○ Hollowness	○ Frontalis	○ Soft palpate movement & uvula
❖ Motor power	○ Temporalis ○ Masseter ○ Pterygoid	○ Orbicularis oculi ○ Buccinators ○ Retractor anguli ○ Orbicularis oris	
❖ Sensory	○ 3 areas in face ○ General sensation of anterior 2/3 of tongue	○ Taste sensation of anterior 2/3 of tongue	○ General & taste sensation in posterior 1/3 of tongue
❖ Reflexes	○ Jaw ○ Palatal	○ Glabellar	○ Pharyngeal ○ Palatal
	○ Corneal (conjunctival)		---

G. CN II

H. CN 12

- | | | |
|--------------|---|------------------------------------|
| ❖ Inspection | Shoulder Drop, head tilted to opposite side | Fasciculations, Wasting, Deviation |
| ❖ Motor | Sternomastoid & trapezius | Power |

4. Cranial meningeal irritation = neck examination

- Neck rigidity, Opithotonus, Lossegue's sign, Kernig's sign, Brudzinski's sign

5. Examination of the motor system

A. Inspection

- | | | | |
|----------------------------------|--------------------------|-------------------------------|------------------------------------|
| ○ Abnormal involuntary movements | ○ Posture of the patient | ○ Circumference of the muscle | ○ Dysplastic changes & Deformities |
|----------------------------------|--------------------------|-------------------------------|------------------------------------|

B. Palpation : muscle tone

- | | |
|---------------------------|---|
| ○ Wrist & ankles | ○ Shaking method |
| ○ Elbows & knees | ○ Passive flexion & extension |
| ○ Shoulder : Gower's test | ○ Hip : rolling & circumduction
○ Hip & knees: passive elevation |

C. Percussion : fasciculations & myotonia

D. Muscle power : without resistance & against resistance

- | | | | |
|---------------------|--------------------------------|----------------------|-----------------------------|
| ○ Shoulder & hip | Flexion, Extension | Abduction, Adduction | Internal, external rotation |
| ○ Elbow & knee | Flexion & extension | | |
| ○ Wrist | Flexion & extension | | |
| ○ Ankle | Planter flexion & Dorsiflexion | | |
| ○ Hands; fingers | Flexors, Extensors | Abductors, Adductors | Opposition, Lumbricles |
| ○ Toes : | Planter flexion & Dorsiflexion | | |
| ○ Abdominal muscles | Flexors & extensors | | |

E. Coordination, repeated 2 times (once with eye opened & the other with eye closed)

- | | | | |
|-----------|------------------|--------------------|---------------------------------|
| ❖ In UL: | ○ Finger to nose | ○ Finger to finger | ○ Finger to fixed doctor finger |
| ❖ In LL : | ○ Heel to knee | | |

6. Reflexes

- | | |
|----------|----------|
| ❖ In UL: | ❖ In LL: |
|----------|----------|

A. Deep reflexes :

- | | |
|---|----------------------------------|
| ○ Brachioradialis or Supinator, Biceps, Triceps | ○ Tendon Knee jerk, Ankle reflex |
|---|----------------------------------|

B. Pathological reflexes : normally absent, if exaggerated they denote UMNL, done only in hyperreflexia only:

- | | |
|--|----------------------|
| ○ Pectoralis, Deltoid, Supraspinatus, Finger flexion, Wetenberg, Hoffman's | ○ Adductor, Patellar |
|--|----------------------|

C. Search for clonus only in hyperreflexia :

- | | |
|----------------|---------------------------------|
| ○ Wrist clonus | ○ Ankle clonus, Patellar clonus |
|----------------|---------------------------------|

○ Superficial reflexes

- | | | | |
|-------------|---------------|-----------------|-----------|
| ○ Abdominal | ○ Cremasteric | ○ Gluteal, anal | ○ Planter |
|-------------|---------------|-----------------|-----------|

7. Examination of sensory system

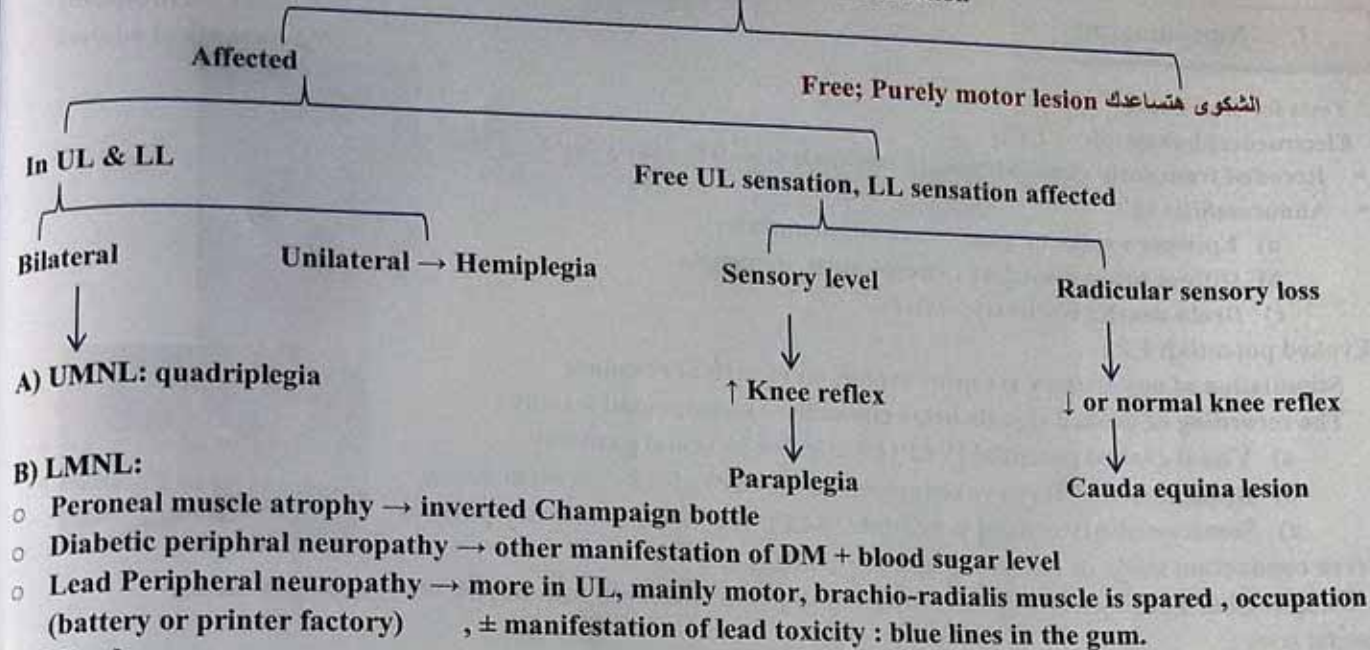
- | | | | |
|----------------------|---|----------------|----------------|
| ○ Superficial : | Pain, touch, temperature | | |
| ○ Deep sensation | joint, Vibration | Muscle & nerve | Romberg's test |
| ○ Cortical sensation | Tactile localization, Tactile discrimination, Stereognosis, Graphosthesia, Perceptual rivalry | | |

8. Examination of others: cranium, back, spine & gait

9. Examination of other systems

SCHEME OF NEUROLOGY

☠ Examine Superficial Sensation



A) Mixed:

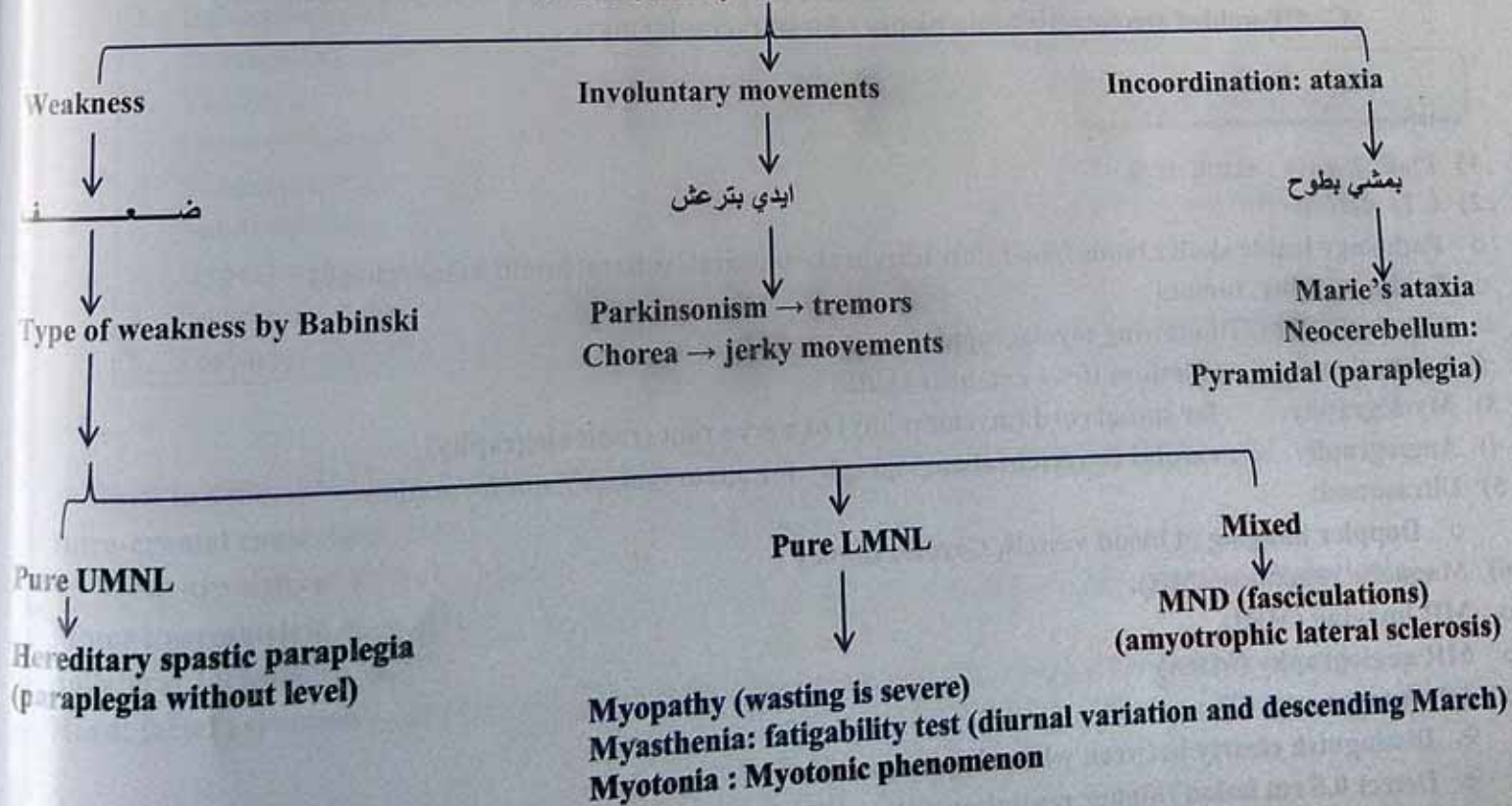
1- SCD: affection of

- { PN & Posterior column → ↓ tone & reflexes } & { Pyramidal tract → ↑ tone & reflexes },
- So lesion depend on which type predominate:
 - Flaccid type : if the PN is the predominant
 - Spastic type : if the pyramidal tract lesion is the predominant

2- Friedreich's ataxia:

- Peripheral neuropathy (Stock & glove hypoesthesia), Archicerebellum, Pyramidal (paraplegia) & Posterior column affection

☞ Free; Purely motor lesion الشكوى هتساعدك



INVESTIGATION OF A NEUROLOGICAL CASE

I. Non-imaging :

A. Tests for functions:

1) Electroencephalography : EEG

- Recorded from scalp electrodes on 16 channels simultaneously for 1 - 3 mm.
- Abnormalities in :
 - a) Epilepsy : spike or spike wave abnormalities
 - b) Diffuse brain disorders : encephalitis, dementia
 - c) Brain death : isoelectric = flat



2) Evoked potentials EP :

- Stimulation of any sensory receptor evokes an electrical response
- The recording of evoked signals helps clinically – unsuspected lesions :
 - a) Visual evoked potential (VEP) : for lesion in visual pathway
 - b) Brain stem auditory evoked potential (BAEP) : for brain stem lesion
 - c) Somato-sensory evoked potential (SSEP): for lesion in sensory pathway



3) Nerve conduction study & electromyography (EMG)

- Using surface needle electrode to record action potentials from muscles or nerves close to skin

B. Special tests :

1) Blood tests:

- A. Antiphospholipid antibodies : diagnose stroke due to hypercoagulable state
- B. Antibodies to acetylcholine receptor ; in myasthenia gravis
- C. Vitamin B12 deficiency in anemia

2) Lumbar Puncture:

- Diagnosis of meningitis & encephalitis
- Measurement of CSF pressure : in ↑ ICT



3) Biopsy:

- A. Muscle biopsy : for muscle disorder
- B. Peripheral nerve biopsy : usually sural nerve at ankle, for diagnosis of polyneuropathy
- C. CT-guided stereotactic brain biopsy : for intracranial mass

II. Imaging :

1) Plain X-rays : skull, neck

2) CT: detect:

- Pathology inside skull : bone, blood (intracerebral, subdural, subarachnoid hemorrhage)
- Brain: atrophy, tumors
- Contrast CT : CT following myelography

→ Limitations : missed lesions if < 1 cm, near skull

3) Myelography : for spinal cord (myelography) or nerve root (radiculography)

4) Angiography : carotid & vertebral angiography for aneurysm, AV malformation

5) Ultrasound:

- Doppler imaging of blood vessels, Carotid duplex

6) Magnetic resonance (MR):

- MR imaging (MRI)
- MR angiography (MRA)

○ Advantages over CT :

- ↳ Distinguish clearly between white & gray matter
- ↳ Detect 0.5 cm lesion (higher resolution), Direct imaging of spinal cord & root
- ↳ No radiation, MRA images blood vessels without need for contrast

→ Limitations : time / cost – claustrophobia

- 7) **Radioisotopes** : assess blood flow & specific areas of brain
- A. Single Photon Emission Computerized Tomography "SPECT"
- B. Position Emission Tomography "PET"
- C. Isotopic scan :
- Isotope brain scan : for intracranial lesions not detected by CT or MRI
 - Isotope brain scan : Technetium 99 – TC 99m for skull & vertebral lesions (metastases)

→ For more details about imaging, ECG, Clinical pathology, see Paraclinical Made easy for the author.

CLINICAL DISCUSSION

❖ What is Glasgow coma scale?

- A scale to assess the conscious level; it depends on 3 parameters :
- Scale 3 deep coma , scale 15 fully conscious & variable levels of consciousness lies in-between

Eye opening		Verbal response		Motor response	
4	Spontaneous	5	Oriented to time & place	6	Obeys commands
3	To speech	4	Confused words	5	Localize pain
2	To pain	3	Inappropriate words	4	Withdraw to pain
1	None	2	Inappropriate sounds	3	Flex to pain
		1	None	2	Extend to pain
				1	None

❖ What are causes of disturbed conscious level (coma)?

- A. Hysterical
- B. Organic (intra-cranial & extra-cranial)

A. Intra-cranial causes	B. Extra-cranial causes
1. Traumatic e.g. Head injury	1) Toxic : morphine, opiate
2. Inflammatory :	2) Hypoxia : anemia
○ Encephalitis , meningitis , brain abscess	3) Ischemic : cardiac arrest
3. Vascular :	4) Endocrinal : addisonian crisis
○ Cerebral hemorrhage, embolism , thrombosis	5) Metabolic :
○ Subarachnoid hemorrhage	○ Hepatic encephalopathy = LCF
○ Subdural hematoma	○ Uremic encephalopathy = renal failure
○ Hypertensive encephalopathy	○ Hypoglycemia coma
4. Neoplastic : primary or secondary	○ Diabetic Ketoacidosis
5. Post-ictal state of epilepsy	○ Diabetic hyperosmolar non ketotic coma

❖ How to differentiate intra from extra-cranial causes of coma ?

- Intra-cranial causes associated with signs of lateralization

❖ What are signs of lateralization?

- Motor : asymmetric deep reflexes, unilateral positive Babinski sign, unilateral hypotonia or hypertonia, , unilateral Jacksonian fits
- Head: facial asymmetry, unequal pupil, Deviation of eyes to one side, tilting of the head to one side

FREQUENTLY ASKED QUESTIONS FOR MID & END ROUND EXAM

Test yourself!

1. What is pyramidal tract pathway ?	2. What is the dermatomal supply of the UL & LL?
3. What are criteria of LMNL?	4. What are criteria of UMNL?
5. What is the nerve supply of tongue?	6. What are the sure signs of bilateral pyramidal tract lesion?
7. What is meant by Gower's test, Gower's sign & Gower's disease?	8. What is grading of muscle power?
9. What is grading of deep reflex?	10. What is inverted supinator reflex?
11. What are causes of Babinski sign?	12. What are causes of equivocal planter reflex?
13. What are causes of hyperreflexia?	14. What are causes of hyporeflexia?
15. What are causes of lost ankle, preserved knee reflex?	16. What are causes of lost ankle, exaggerated knee reflex?
17. What are sure signs of pyramidal tract lesion?	18. What are causes of hypotonia?
19. Pattern of sensory loss	20. Gait in pyramidal tract lesion & other gaits you know
21. What are the differences between rigidity & spasticity ?	
22. What is the root value of the following reflex: ○ Biceps, triceps, Brachioradialis , knee, ankle, adductor, patellar, jaw, glabellar, corneal & conjunctival, pharyngeal, palatal , planter?	
23. All questions answered in the clinical discussion are very important.	

O.S.C.E QUESTIONS

▪ Examine for pyramidal tract lesion in LL	▪ Examine motor part of facial nerve & deep sensation of body
▪ Examine deep & cortical sensation	▪ Examine 3, 4, 6 cranial nerves
▪ Search for cerebellar lesion & superficial sensation	▪ Deep reflexes of UL
▪ Examine trigeminal nerve & deep sensation of LL	▪ Reflexes of LL
▪ Examine motor system of lower limb (hemiplegia , paraplegia)	▪ Examine motor system of both LL "reflexes not required"
▪ Examine motor system lower limb (deep reflex not required)	▪ Examine motor power of both LL
▪ Power and tone of LL	▪ Reflexes of both upper limbs
▪ Examine motor system of both UL	▪ Examine cranial nerves (5th, 7 th , 12 th)

Hemiplegia

❖ Level of the lesion:

A. Cerebral	B. Brain stem	C. Spinal cord
○ Cortical: coma or convulsions, aphasia ○ Subcortical: more extensive, coma ○ Capsular: 99% of our cases	○ Crossed hemiplegia: CN palsy on opposite side of the hemiplegia	○ Brown Sequard syndrome: no CN affection

❖ Evidence of cerebral hemiplegia:

- 1) Cranial nerve lesion → not spinal
- 2) Not crossed hemiplegia → not brainstem

❖ Characters of capsular hemiplegia:

- 1) No coma, convulsion & aphasia → not cortical
- 2) Complete hemiplegia associated with UMN facial & hypoglossal paralysis on opposite side of the lesion
- 3) Complete hemi-hypoesthesia on opposite side of the lesion

❖ Etiology:

- 1) **Vascular**: 99% of our cases are due to vascular cause (stroke)
 - Acute onset & regressive course
 - The most common cause
- 2) No fever or meningeal irritation → not inflammatory
- 3) No history of trauma → not traumatic
- 4) Not gradual onset & progressive course → not neoplastic
- 5) No remittent course → not demyelination

❖ Type of vascular lesion:

	A. Embolism	B. Thrombosis
History		
Personal	Young, middle age	Old age
Past	Rheumatic fever	HTN, DM
HPI (Onset)	Seconds	Hours
Associations	Dyspnea (MS) Palpitation (AF)	Anginal chest pain (atherosclerosis of the coronaries)
Examination		
Pulse	Irregular (AF)	regular
Blood pressure	Normal	HTN
Heart	Mitral stenosis murmur	Ischemic heart disease
N.B.: Hemorrhage: early death		

❖ Which artery is occluded and why? According to presentation:

1. Capsular branch of Middle Cerebral Artery "lenticulo striate":

- The only one which gives complete hemiplegia (UL = LL)
 - The commonest to be occluded as its direct continuation of carotid artery
2. Main branch of MCA: UL > LL, aphasia is present
 3. Capsular branch of ACA: monoplegia only
 4. Main branch of ACA: LL > UL - mentality changes are present
 5. Capsular branch of PCA: thalamic syndrome
 6. ICA: Ipsilateral blindness, absent carotid pulsation (palpated in tonsillar fossa)

❖ Stage:

A. Stage of neuronal shock	B. Stage of neuronal recovery	C. Stage of spasticity
○ Complete paralysis	○ Mild improvement in power	○ Marked improvement in power (not distal)
○ Lost tone, deep reflexes	○ Reappearance of tone, deep reflexes	○ Hypertonia, hyper-reflexia

❖ **Signs of pyramidal tract lesion; organic lesion:**

- Clonus, Positive Babinski sign, Positive pathological reflexes, Hyper-reflexia & Hypertonia

❖ **Differential diagnosis from hysterical by:**

- Side gait test
- Babinski sign
- Hoover sign:

📌 **Remember : 2 Hoover sign :**

1. Chest : Inspiratory indrawing of costal margin in COPD due to low flat diaphragm (See before)
2. Neurology: in hemiplegia to differentiate between organic and hysterical causes

طريقة الفحص : نيم العيان على السرير

(2) خلي العيان يعمل بالرجل السليمة flexion to the hip against resistance	(1) خط إيدك تحت كعب الرجل المريضة واطلب منه بضغط بها على إيدك
	
النتيجة: لو محسّش بضغط على إيدك من الرجل المريضة في المرة الأولى وحسيتها في الثانية تبقى الحالة hysterical ويكده تبقى Positive Hoover's sign لو محسّش بضغط على إيدك في الحالتين تبقى الحالة organic	

❖ **Gait:** circumduction gait

❖ **What is the best investigation in hemiplegia? CT**

❖ **What is the treatment of hemiplegia?**

- Care of comatose, Physiotherapy
- Treatment of the cause: (CVS causes)
- Embolic cases → anti-coagulant
- Thrombotic cases → antiplatelet

❖ **N.B.:** Any patient with stroke you must give special attention to carotid artery by :

- Examination : pulse
- Investigation : Doppler, Duplex, MRA

❖ **Example of diagnosis :**

- Organic right hemiplegia, it is embolic at the capsular level, the patient is at the spastic stage

**Brown Sequard syndrome
Spinal hemiplegia
(Hemi-section of spinal cord)**

❖ **Definition :** lesion is on one side of the cord & it's situated between C1 & C5 segments

❖ **Etiology :** traumatic : stab wound , disc prolapse

❖ **Clinical picture :**

1. Hemiplegia on the same side of the lesion
- ↑↑↑ tone, deep reflexes , +ve Babinski & Weakness of Δ tract distribution
2. Sensory ataxia on the same side of the lesion : Posterior column affection, deep sensory loss
3. Superficial sensory loss on the opposite side of the lesion : Pain , temperature sensation
4. No cranial nerve affection

❖ **Investigations:** CT, MRI

❖ **Treatment :** as hemiplegia but no need for care of comatose patient & medication given for vascular cau

Capsular hemiplegia, Thrombotic

- ❖ **Personal history:** A known diabetic and hypertensive patient, ... (name) ... male patient, 45 years old, driver, from Cairo, has 3 off spring, the youngest is ten years old, heavy smoker, Rt. handed.
- ❖ **Complaint:** Heaviness on Rt. upper and lower limbs of 2 years duration.
- ❖ **HPI:**
 - The condition started 2 years ago and reached its complete picture within hours. This onset was not associated with fever, trauma, vomiting, coma or convulsion. The patient at first developed **complete paralysis** associated with **flaccidity, tingling and numbness** in the Rt. upper and lower limbs and also had a **speech defect**.
 - 6 weeks later, the patient experienced **weakness of gradual onset** associated with **stiffness with no wasting or twitches**. This weakness affected the Rt. upper and lower limbs affecting **distal** more than proximal muscles, **abductor** more than adductor muscles, **extensor** more than flexors muscles in **Rt. upper limb** and **flexor** more than extensor muscles in **Rt. lower limb**. No involuntary movements. The condition was associated with **loss of sensation** in Rt. Upper and lower limbs and the patient felt as if his Rt. leg **walks on cotton** and there is **marked improvement in speech**.
 - Apart from **accumulation of food** behind Rt. cheek, **dripping of saliva** from Rt. side of mouth, **deviation of mouth** to Lt. side, **deviation of tongue** to the Rt. Side and **dropping of the Rt. Shoulder**, There are no symptoms suggesting other cranial nerve affection.
 - No symptoms of **sphincteric** affection, No symptoms of increased **I.C.T.**
 - No symptoms of **cardio vascular system** affection, No other system affection
- ❖ **Past history:**
 - Diabetes started 8 years ago and manifested by polyphagia, polyurea, polydypsia and treated by dimicron.
 - HTN started 6 years ago and manifested by headache blurred vision and treated by capozid.
 - No past history of fever, operation, other drug intake.
- ❖ **Family history :**
 - No similar condition in family. No consanguinity, No common disease in family.
- ❖ **General examination :**
 - **Temperature:** 37° c.
 - **Blood Pressure:** 130/70 (may be higher).
 - **Pulse:** regular, 80 beat/minute, average volume, no special character, vessel wall not felt, equal in both sides with intact peripheral pulsation.
 - **Mentality:** The patient is fully conscious, well oriented for time, place and person. Average mood and memory. The patient is co-operative with average intelligence.
- ❖ **Local examination :**
- ❖ **Examination of speech : no aphasia , no dysarthria**
- ❖ **Cranial nerve examination :**
 - **Facial nerve : by inspection**, there is obliterated right nasolabial fold but symmetrical forehead, no tears, no dripping of saliva, no deviation of the mouth. **By exam of power**, there is deviation of angle of mouth to left side, The patient can not blow his right cheek and can't whistle but can close his eyes firmly with normal elevation of both eye brows and symmetrical forehead wrinkles on both sides..
 - **Hypoglossal nerve :** the tongue is deviated to the Rt. side, decrease power on Rt. side but no fasciculation or wasting.
 - **Accessory nerve:** there is affection of trapezius of Rt. Side
 - **Trigeminal nerve:** loss of sensation on Rt. side of face.
 - Other cranial nerves were examined but no abnormality detected
- ❖ **Examination of motor system :**
 - **By inspection:**
 - ❖ There is flexion of Rt. upper limb at elbow, extension in Rt. lower limb at knee.
 - ❖ No skeletal abnormalities, no trophic changes, no involuntary movement no muscle wasting
 - **By palpation:**
 - ❖ There Is normal tone in It upper and lower limbs, in Rt. Upper and lower limbs there is hypertonia in the form of spasticity affecting antigravity muscle indicating UMNL in the Rt. upper and lower limbs.
 - **By percussion:** No fasciculation or myotonia.

Examination of muscle power:

- There is normal muscle power in Lt. upper and lower limbs, in Rt. limbs: there is weakness affecting distal more than proximal, abductors more than adductors, in the Rt. Upper limb extensors are weaker while in the Rt. Lower limb flexors are more weaker. Indicating weakness of Δ tract distribution in the Rt. Upper and lower limbs.
- N.B: power of abdominal muscles showed no abnormality.

Coordination :

- Cannot be examined on Rt. side because of weakness, on Lt. side there is normal coordination proved by finger to nose, finger to finger in left UL in both eye opening and eye closure and finger to doctor's finger only in eye opening and heel to knee to left LL in both eye opening and eye closure.

Reflexes:

- There is normoreflexia in Lt. upper, lower limbs.
- In Rt. upper limb: hyperreflexia with +ve pathological reflexes.
- In Rt. lower limb: hyperreflexia with +ve pathological reflexes and +ve clonus.
- +ve babinski on Rt. side and normal planter response on the left side.
- Abdominal reflexes lost on the right side.

- Sensory:** complete hemi-hypoesthesia on Rt. side.

- Back :** no deformities, no pigmentation

- Gait:** circumduction gait

No affection in other system examination

- Diagnosis:** Organic Rt. sided hemiplegia. It is thrombotic at capsular level. The patient is in the spastic stage.

Why Hemiplegia?

Unilateral UMNL:

- Tone antigravity + spasticity.
- Power distribution, pronator, abd.
- Hyper reflexia + pathological reflexes + clonus.
- +Ve babinski.
- Hypoglossal (no fasciculation, wasting) and UMNL facial n. palsy.
- Complete hemi hypoesthesia.

Why Capsular?

Motor: complete hemiplegia.

Sensory: complete hemi hypoesthesia.

Not cortical: no convulsion, coma, aphasia. Usually (monoplegia)

Not spinal: cranial N affection excludes spinal.

Not brainstem: not crossed hemiplegia,

How would you proceed to manage this case?

Investigation : CT, MRI, carotid MRA, cardiac.

Treatment: Physiotherapy, Vitamins, Antiplatelet drugs (thrombosis), anticoagulant (embolic)

Capsular hemiplegia, Embolic

Differs from thrombotic hemiplegia in the following :

A. History:

- The patient is **younger** at time of stroke.
- The condition reached its complete picture within **seconds**.
- Start with : the condition started Years ago by gradual onset, progressive course of exertional dyspnea. At first, it was on climbing the 2nd floor then it became progressively increased till became at rest and the patient had orthopnea he used to sleep on 3 pillows and he had paroxysmal nocturnal dyspnea he waked up after 2 hours of sleep with dyspnea, cough and wheeze .after 15 minutes, relief usually occurred. This dyspnea was associated with rapid irregular palpitation increased with exertion then afteryears the patient developed like thrombotic
- Past history of **rheumatic fever**

B. Examination:

- Pulse** may be irregular, **Blood pressure** is usually normal, **Cardiac exam** may reveal signs of M.S (may be with AF)

Spinal hemiplegia

- ❖ **Personal history:** ...
- ❖ **Complaint:** Heaviness on Lt. upper and lower limbs of 6 years duration.
- ❖ **HPI:**
 - The condition started 6 years ago by a car accident. The patient at first had **complete flaccid paralysis** associated with **tingling and numbness** in the Rt. Upper and lower limbs.
 - 2 weeks later, the patient experienced **weakness** of gradual onset and progressive course associated with **stiffness** with **no wasting or twitches**. This weakness affected the Lt. upper and lower limbs affecting **distal** more than proximal muscles, **abductor** more than adductor muscles, **extensor** more than flexor muscles in Lt. upper limb and **flexor** more than extensor muscles in Lt. lower limb. No involuntary movements.
 - The condition was associated with **loss of sensation** in Rt. Upper and lower limbs and the patient felt as if his Rt. leg **walks on cotton**.
 - No symptoms of **Cranial nerve** affection.
 - No symptoms of **speech** affection.
 - No symptoms of **sphincteric** affection.
 - No symptoms of increased **I.C.T.**
 - No **other system** affection
- ❖ **Past history:** No History of D.M, HPN, drugs or operations.
- ❖ **Family history:**
 - No similar condition in family.
 - No consanguinity. No common diseases in family.
- ❖ **General examination:**
 - **Temperature:** 37° c.
 - **Bl. Pressure:** 120/80.
 - **Pulse:** regular, 70 beat/minute, average volume, no special character, vessel wall not felt, equal in both sides with intact peripheral pulsation.
 - **Mentality:** The patient is fully conscious, well oriented for time, place and person. Average mood and memory. The patient is co-operative with average intelligence.
- ❖ **Local examination:**
- ❖ **Examination of speech :** normal
- ❖ **Examination of CN:** normal
- ❖ **Examination of motor system:**
- ↳ **Inspection:**
 - There is flexion of Lt. upper limb at elbow, extension in Lt. lower limb at knee.
 - No skeletal abnormalities, no trophic changes, no involuntary movement, no muscle wasting.
- ↳ **Examination of Tone:**
 - There is normal tone in Rt. upper and lower limbs.
 - In Lt. Upper and lower limbs there is hypertonia in the form of spasticity affecting antigravity muscle (indicating UMNL in the Lt. upper and lower limbs).
- ↳ **Percussion:** No fasciculation or myotonia.

❖ Examination of muscle power:

- There is normal muscle power in Rt. upper and lower limbs.
- In Lt. upper and lower limbs: there is weakness affecting distal more than proximal muscles, abductor more than adductor muscles, in the Lt. Upper limb extensors are weaker while in the Lt. Lower limb flexors are more weaker (Indicating weakness of Δ tract distribution in the Lt. Upper and lower limbs).

❖ Coordination:

- Cannot be examined on Lt. side because of weakness
- On Rt. side there is normal coordination proved by finger to nose, finger to finger, finger to doctor's finger in both eye opening and eye closure.

❖ Reflexes:

○ Deep reflexes

- There is normoreflexia in Rt. upper, lower limbs.
- In Lt. upper & Lower limbs: hyperreflexia with +ve pathological reflexes.

○ Superficial reflexes

- +ve Babinski on Lt. side and normal planter response on the Rt. side.
- Abdominal reflexes: lost on the left side.

❖ Sensory :

- **Superficial sensation:** Right sided hemi-hypoesthesia (not complete i.e. face is spared).
- **Deep sensation:** Deep sensory loss in Lt. upper and lower limbs i.e. lost muscle, nerve, vibration, joint sense with +ve romberg's test. (Post. Column affection).
- **Cortical sensation:** can't be examined.

❖ Investigation : CT, MRI

❖ Treatment : physiotherapy, vitamins

❖ **Diagnosis : Organic Lt. sided hemiplegia. It is traumatic at spinal level. The patient is in the spastic stage.**

❖ **Why Hemiplegia?** (Unilateral UMNL)

1. Tone spasticity in antigravity muscles.
2. Power distribution of weakness.
3. Hyper reflexia + pathological reflexes.
4. +Ve Babinski.

❖ **Why Spinal?**

- History of **trauma**.
- **No Cranial Nerve** affection.
- **Hemiplegia** on the same side on the lesion.
- Contralateral Hemi-**hypoesthesia**.
- Ipsilateral **deep sensory** loss.

Paraplegia

❖ Level of the lesion :

A. Spinal cord lesion		
○ Focal , systemic or disseminated	B. Cerebral lesion	C. Brain stem lesion
	○ ↑ ICT, Coma, convulsions	○ CN affection

❖ No ↑ ICT, Coma, convulsions → not cerebral lesion

❖ No CN affection → not brainstem lesion

❖ So our case is , **Spinal cord lesion**

❖ Causes:

1. With a level (focal):

- Acute onset, regressive course → inflammatory e.g. Transverse myelitis
- Vascular as anterior spinal artery occlusion → posterior column is preserved
- Gradual onset , progressive course → **compression** : either extra – or intramedullary block

Causes		
A. Extra-medullary block		B. Intra-medullary block
○ Vertebral compression as trauma, tumor , degenerative , deformities , Pott's disease (TB) ○ Meningeal compression e.g. meningioma		○ Cord compression as syringomyelia , glioma, ependymoma of the cord
❖ History		
Duration	○ Long	○ Short
Onset	○ Painful (posterior root irritation)	○ Painless
❖ Clinical picture		
Motor	○ Usually asymmetrical	○ Usually symmetrical
Sensory	• Sensory level • All types of sensation are diminished below level are including pain, temperature & touch • Early loss of sensation in the saddle area	• Jacket sensory loss • Dissociated sensory loss i.e. loss of pain & temperature with preservation of touch • Late loss of sensation in the saddle area (sacral spare)
Bladder	○ Late disturbance	○ Early disturbance
❖ Investigations		
C.S.F.	○ Marked lowering of pressure ○ Complete dynamic block ○ Froin's syndrome : spontaneous coagulation, xanthochromia, cyto-albuminous dissociation	○ Less marked lowering of pressure ○ Partial or no dynamic block. ○ No Froin's syndrome
Plain x-ray	○ Possible vertebral lesion.	○ Normal x-ray.
Myelography	○ Saddle shaped block.	○ Fusiform shape

2. Without a level: (Systemic & Disseminated):

A. Systemic :	B. Disseminated :
• HSP (Hereditary Spastic Paraplegia). • Pellagra & SCD	• Remission & exacerbation: D.S. (disseminated Sclerosis), disseminated encephalomyelitis (DEM)

❖ Stage of paraplegia : flaccid or spastic

A. Stage of Flaccidity	B. Stage of Spasticity :
○ Stage of neuronal shock ○ Immediately follow lesion ○ Complete hypotonia & hyporeflexia of acute onset = LMNL like ○ Remain 2-6 weeks	❖ Signs of pyramidal tract lesion; bilateral UMNL: ○ Paralysis of paresis of both LL ○ Hypertonia (clasp knife) ○ Hyper-reflexia with or without clonus ○ Positive Babinski ○ Precipitancy

❖ In spastic stage : is it in extension or flexion

	A. Paraplegia in extension	B. Paraplegia in flexion
Cause	○ Pyramidal lesion	○ Pyramidal & extrapyramidal
Timing	○ After acute shock stage or gradual onset from start	○ After acute shock stage or after extension stage
Hypertonia	○ More in extensors صعوبة في التثني	○ More in flexors صعوبة في الفرد
LL	○ Extended رجله مفروده في السرير	○ Flexed رجله متنيه في السرير
Deep reflexes	○ Exaggerated	○ Less
Clonus	○ Present	○ Absent
Mass reflex	○ Absent	○ Present
Bladder	○ Precipitancy	○ Automatic bladder

❖ **Pierre Marie Foix test:** to detect paraplegia passing from extension to flexion :

☞ Firm passive planter flexion of toes & foot → spontaneous withdrawal reflex i.e. flexion of hip, knee & dorsiflexion of ankle

❖ **Mass reflex :** in paraplegia in flexion :

☞ Scratching skin over medial side of thigh → spontaneous urination, defecation & sweating

☞ Squeezing glans penis → reflex erection & ejaculation

❖ **Gait in paraplegia :** scissoring gait

❖ **Level of the lesion:**

- History level : root pains; girdle pain (site of pain indicates site of compression)
- Motor level (Beevor's sign)
- Reflex level (abdominal reflexes)
- Vertebral level (راجع الجدول)
- Sensory level (الأهم في الإمتحان)

❖ **Example of diagnosis :**

- Organic paraplegia of spinal focal type. It's due to extra-medullary lesion at the level of T10.
- The patient is in the spastic stage (in extension).

Paraplegia

❖ **Personal history**

❖ **Complaint:** Heaviness of both lower limbs of 22 years duration.

❖ **HPI:**

- The condition started 22 years ago when he fell from a height of 3 meters on his back. At 1st the patient suffered from complete **paralysis of both lower limbs with flaccidity and girdle pain** at the level of the umbilicus increased by work, coughing, sneezing relieved by rest. Then the patient was admitted to hospital and investigated by X-ray with contrast and Exploratory operation was done.
- 4 weeks later, the patient experienced gradual onset, progressive course of **weakness** associated with **stiffness**, with **no wasting or twitches**. This weakness was **distal** more than proximal, in the **abductor** muscles more than adductors, in **flexor** more than extensors with no involuntary movements. This weakness was associated with **diminished sensation** in both lower limbs and the patient felt as if he **walks on cotton**.
- No symptoms of **sphincteric** affection.
- No symptoms of increased **I.C.T.**, No symptoms of **speech** disorders.
- No symptoms suggesting other system affection.

❖ **Past history:**

- No past history of fever, bilharziasis, drugs or operations.

❖ **Family history:**

- No similar condition in family, No consanguinity.
- No common disease in family

❖ **General examination:**

- **Temperature:** 37.2° c.
- **Bl. Pressure:** 130/70.
- **Pulse:** regular, 70 beat/minute, average volume, no special character, vesselwall not felt, equal in both sides with intact peripheral pulsation.
- **Mentality:** The patient is fully conscious, well oriented for time, place and person. Average mood and memory. The patient is co-operative with average intelligence.

❖ **Local examination:**

❖ **Examination of Speech:** Normal.

❖ **Examination of Cranial Nerves:** Normal.

❖ **Examination of motor system :**

↳ **Inspection**

- There is extension in both lower limbs (paraplegia in extension).
- No skeletal deformities, no trophic changes, no involuntary movement no muscle wasting.

↳ **Examination of tone**

- There is normal tone in both upper limbs .
- Bilateral asymmetrical Hypertonia in lower limbs in the form of spasticity affecting antigravity muscles. It is more on Lt. lower limb (suggesting UMNL).

↳ **Percussion**

- No fasciculation or myotonia.

↳ **Examination of muscle power**

- There is normal muscle power in both upper limbs,
- Bilateral asymmetrical Weakness in lower limbs. It is distal more than proximal, abductors more than adductors, flexors more than extensors. It is more on Lt. lower limb.

↳ **Coordination**

- Coordination cannot be examined on both lower limbs because of weakness.
- Normal coordination in both upper limbs confirmed by finger to nose, finger to finger, finger to doctor's finger in both eye opening and eye closure.

❖ Reflexes

- There is normoreflexia in both upper limbs.
- In Both lower limbs: hyperreflexia with +Ve pathological reflexes (Patellar, Adductor), no ankle or patellar clonus.
- +ve Babinski on both sides.
- Abdominal reflex : lost below the level of the umbilicus .

❖ Sensory

- Superficial sensations: Sensory Level at T10.
- Deep sensation: lost.
- Cortical sensation : can't be examined in both lower limbs.

❖ Back Scar of exploration at the level of T7.

❖ Gait Scissoring gait.

❖ No affection of other system examination

❖ Investigation : Plain X-Ray , Myelography .

❖ Treatment : physiotherapy

❖ Diagnosis :

- Organic Paraplegia Of Spinal Focal Compression Type. It's due to Extramedullary lesion at the level of T10. The patient is in the spastic stage (in Extension).

❖ Why Paraplegia? (UMNL Bilateral Δ) : Affect both L.L. + Organic

- Hypertonia in antigravity + spasticity.
- Muscle Weakness: distal, progravity, abductors.
- Hyperreflexia + pathological reflexes.
- +Ve Babinski in both L.L.
- Sensory Level at umbilicus.
- No cranial nerve affection.

❖ Why Spinal?

- Not Cortical: Rare, Need parasagittal lesion, no coma convulsion or aphasia.
- Not brainstem: no affection of Resp. Center, no Cranial N. involvement, need lesion in midline to affect L.L which is medial and no bladder dysfunction.

❖ Why Focal?: Level .

❖ Why Compression?: No history of fever (inflammation), +Ve history of trauma.

❖ Why Extra-medullary?

- Sensory Level.
- Asymmetrical Power, reflexes.
- Girdle pain.
- No Sphincteric affection.

❖ Why Level of T10?

- History: girdle pain, exploration at T7.
- Sensory level at T10.
- Abdominal Reflex lost at T10.
- Vertebral :scar.

❖ Why Spastic Stage:?

❖ Hypertonia

❖ Hyperreflexia

❖ Why Extension? Patient's Position.

❖ N.B.:

A. Inflammatory paraplegia differs from traumatic one in the following :

- Fever at the onset.
- Regressive course.
- Corticosteroids in TTT.

B. Retention of urine may occur at the onset of acute lesions.

Peripheral neuropathy

❖ **Clinical picture of polyneuropathy:**

❖ It will present by the 3 following manifestations in difference combinations:

A. Motor:

1. Weakness or paralysis of L.M.N. nature (wasting, hypotonia, hyporeflexia . . .).
2. The weakness and wasting are:
 - Bilateral and symmetrical.
 - Affecting L.L. more than U.L.
 - Affecting distal muscles more than proximal muscles.
 - Affecting extensors more than flexors, affecting adductors more than abductors
3. The weakness in the extensors of the distal group of muscles leads to bilateral foot drop and wrist drop.
4. The ankle reflex is lost while the knee reflex is preserved.
5. The cranial nerves may be affected specially CN 3, 6, 7 and 10.
6. The gait is high steppage due to the foot drop.

B. Sensory:

1. History: pain and paraesthesia in the limbs, specially distally.
2. Examination:
 - Superficial sensory impairment of the stock and glove nature.
 - Deep sensory loss specially distally with absence of deep reflexes.

C. Autonomic:

1. Vasomotor: coldness and cyanosis of the limbs.
2. Cutaneous: loss of hair, brittle nails, trophic ulcers.

❖ N.B.: Regardless of the cause of the polyneuropathy, the clinical picture is essentially the same; variations depend on whether the motor, sensory or autonomic features predominate.

→ Clinical case of peripheral neuropathy :

Diabetic peripheral neuropathy

Peroneal muscle atrophy

Diabetic peripheral neuropathy

❖ **Neurological complications of DM:**

1. Peripheral neuropathy : stock & glove hypesthesia & deep sensory loss

2. Brain:

- | | | |
|--|---------------------------|--|
| ○ Cerebro-vascular accidents (stroke). | ○ Coma of different types | ○ Infection: fungal (rhinocerebral mucormycosis) |
|--|---------------------------|--|

3. Brain stem: Argyl Robertson pupil.

4. Spinal Cord: (very very very rare)

- Δ tract affection → Diabetic lateral Sclerosis { Planter reflex may be equivocal due to superficial sensory loss}
- Posterior column affection → sensory ataxia, Diabetic pseudotabes.
- Diabetic SCD.

5. Root: Diabetic Radiculopathy.

6. Muscle: Diabetic amyotrophic:

- Wasting, weakness of quadriceps muscle (proximal muscle!) due to femoral neuropathy.
- Reversible condition improve by diet, exercise, insulin therapy.

❖ **Effect of DM on other systems (FOR MORE DETAILS SEE ENDOCRINOLOGY CHAPTER)**

- | | |
|--|---|
| <ol style="list-style-type: none"> 1. General : <ul style="list-style-type: none"> ○ Pulse : Sinus tachycardia, may be absent peripheral pulsations ○ HTN, Postural hypotension 2. CVS: <ul style="list-style-type: none"> ○ IHD, HTN, Cardiomyopathy 3. Chest : TB follows diabetes like its shadow 4. Abdomen : Fatty liver, Diabetic nephropathy | <ol style="list-style-type: none"> 5. Skin: <ul style="list-style-type: none"> ○ Infection ○ Ischemic (vascular) effect. ○ Neuropathic (P.N) effect. ○ Necrobiosis lipoidica diabetocorum. ○ Bullosus diabetocorum. ○ Diabetic Dermopathy. ○ Effect of TTT : Lipodystrophy & Injection sites |
|--|---|

❖ Autonomic neuropathy in DM:

→ Pathogenesis: Sorbitol Pathway

→ Symptoms & signs:

1. Cardiovascular system:

- Absent respiratory sinus arrhythmias.
- Persistent sinus tachycardia (examine with pulse & ask about palpitation)
- Painless myocardial infarction.
- Postural hypotension (examine with vital sign)

2. Genitourinary:

- Bladder disturbances (incontinence → ask for it)
- Impotence (psychic and organic ask for it)

3. Gustatory sweating (Marked sweating specially with meals → ask for it)

4. Gastrointestinal:

- Gastroparesis diabeticorum (ask for dyspepsia).
- Diabetic enteropathy (ask for nocturnal watery diarrhea and constipation).

Peroneal muscle atrophy

❖ Incidence :

- Positive family history (heredo-familial)
- Appears at 1st at 2nd decade.
- Male > female

❖ Clinical picture:

A. Generally:

1. Bilateral, symmetrical.
2. Gradual onset.
3. Very very very slowly progressive course.
4. Appears in LL → then progress to U.L.



(لكن بعد كثير جداً علشان كده تلاقي ال LL أكثر من ال UL في كل حاجة)

B. Local manifestations:

1. Skeletal deformity.

- Pes cavus, talipes equinus, Hammer toe, ± Kypho-scoliosis.

2. Sensory changes:

- Superficial sensation (stock, glove nature).
- Deep sensation (lost all deep sensation).

3. Motor changes:

→ Muscle state: Wasting (marked, bilateral, symmetrical) which may lead to:

- Pes cavus
- Start in lower limbs in the peronei muscles then anterior tibial group then ascend to involve the muscles of lower 1/3 of thigh resulting in Inverted Champaign bottle appearance.

→ Trophic changes:

- Loss of hair.
- Brittling of nails lusterless.

→ Posture:

- Foot drop (gait)
- Wrist drop.

→ Muscle tone:

- Hypotonia (frog position)

○ Power:

- Mild disturbance of motor power i.e. Discrepancy between degree of wasting, degree of weakness

○ Reflexes:

- Lost ankle, preserved knee (early).

❖ Associated signs:-

- Degeneration of cerebellum → Friedreich's ataxia.
- Degeneration of AHCs → fasciculations.
- Degeneration of Δ Tract → + ve Babinski.

للـ ال Babinski ممكن يكون موجود فعلاً وما يبانش لو عنده skeletal deformities of big toe

🔑 N.B All data of C/P are a must ± Association

❖ What are other Nomenclature of this disease ? ده هام ممكن تتسأل السؤال ده

1. Peroneal muscle atrophy.
2. Charcot-Marie-Tooth disease. اسم ثلاث علماء اشتتركوا في وصفه
3. Hereditary Sensory Motor Neuropathy type I (HSMNI).

Diabetic peripheral neuropathy

❖ Personal history :....

❖ **Complaint:** Loss of sensation in both hands and feet of 15 years duration.

❖ HPI:

- The condition started 15 years ago by nocturnal **burning pain** associated with **tingling, numbness** started in both feet then progressed, one year later, to involve both hands then the patient developed gradual **loss of sensation** in both hands and feet, and he felt as if he **walked on cotton**
- 4 years later, the patient experienced **weakness** associated with **flaccidity, falling of hair, brittle nails** with no **wasting or twitches**. This weakness started in L.Ls then progressed, one year later, to involve both U.Ls. It's more in distal than proximal muscles, in extensor more than flexor muscles, in adductor more than abductor muscles. The patient also suffers from **unsteadiness during eye closure** with no involuntary movements.
- The condition was associated with **diminution of vision, visual field defects, disturbance of color vision, ptosis** in both eyes for which the patient was investigated and treated by laser photocoagulation more than once. The patient **can't close his eyes firmly, with accumulation of the food behind both cheeks**, No symptoms of other cranial nerve affection
- The patient has **organic impotence** with lost morning erection with no history of drugs known to cause erectile dysfunction.
- The patient developed **unsteadiness during standing with palpitation, nocturnal diarrhea, gustatory sweating and dyspepsia**
- No symptoms of increased I.C.T., No speech disturbance., No symptoms suggestive of other system affection

❖ Past history:

- There is past history of **D.M** started 20 years ago manifested by polyuria, polydipsia, polyphagia. The patient is on insulin treatment and his blood sugar is out of control.
- There is past history of **HTN** started 15 years ago manifested by headache, blurred vision. The patient is on capoten and his hypertension is not controlled.
- Appendectomy operation was done at the age of 20 years.
- No history of other drug intake.

❖ **Family history:** No similar condition in family, No consanguinity. No common disease in family

❖ General examination

- **Temperature:** 37.2° c.
- **BL Pressure:** 140/80 (Recumbent position), 100/60 (standing position).
- **Pulse:** regular, 110 beat/minute, average volume, no special character, vessel wall not felt, equal in both sides with absent dorsalis pedis, anterior and posterior tibial and popliteal pulsation with intact femoral, radial, brachial and axillary pulsation.
- **Mentality:** The patient is fully conscious, well oriented for time, place and person, Average mood and memory. The patient is co-operative with average intelligence.
- **Head:** Examine for Retinopathy, teeth (Artificial teeth).
- **LL:** Trophic ulcer, diabetic dermopathy.

❖ Local examination :

❖ Sensory :

❖ **Superficial sensations:** above knee and elbow level stock and glove anesthesia. Circumferential comparison must be done to exclude diabetic radiculopathy.

❖ Deep sensation:

- Joint sense lost on both sides.
- Vibration sense lost at level of peripheral nerve (medial malleolus, radial styloid process) with intact vibration sense at the level of posterior column (ASIS, clavicle).
- Muscle sense lost (Calf muscles).
- Lost nerve sense (Ulnar and lateral popliteal nerves).
- +Ve Romberg's test.

❖ **Cortical sensation:** can't be examined due to loss of superficial sensation.

❖ **Examination of speech : normal**

❖ **Examination of cranial nerve:**

- **Optic Nerve** is affected in the form of: diminution of visual acuity (Rt. eye: can count fingers at one meter, Lt. eye: blind), Tubular visual field defect.
- **Ocular nerves**
 - **Inspection:** bilateral ptosis (thumb test » can't elevate his eye lids), pupils are dilated and irreactive to light or accommodation with no squint.
 - **Power:** loss of eyeball movements in all direction denoting paralysis of recti and oblique muscles of the eye.
- **N.B:** nystagmus and conjugate eye movements can't be examined because of loss of eye movements on examining each eye separately.
- **Reflexes:** absent light and accommodation reflexes.
- **Facial nerve**
 - **Inspection:** symmetrical forehead, obliterated nasolabial folds on both sides with no tearing, no dripping of saliva, no mouth deviation
 - **Power:** patient can't close his eyes firmly, can't elevate his eye brows, can't whistle, can't show his teeth, can't blow his cheeks
 - **Reflexes:** absent glabellar reflex → bilateral LMNL
- ❖ **Examination of motor system :**
- ↳ **Inspection**
 - There is wrist and ankle drop, trophic ulcer in L.L, loss of hair and brittle nail in U.L, L.L.
 - No muscle wasting, no skeletal deformities, no involuntary movement.
- ↳ **Examination of tone**
 - Bilateral symmetrical hypotonia in both upper and lower limbs
- ↳ **Percussion:** No fasciculation or myotonia.
- ↳ **Examination of muscle power**
 - Bilateral symmetrical Weakness in both LIMBS but it is in LL more than UL, It is distal more than proximal, extensors more than flexors., in adductors more than abductors
 - Abdominal muscles: weakness may be attributed to trunkal neuropathy or related to myopathy as the patient gives history of thyrotoxicosis.
- ↳ **Coordination**
 - Cannot be examined on both upper and lower limbs because of weakness.
- ❖ **Reflexes**
 - **Deep reflexes:** Areflexia in both upper and lower limbs.
 - **Superficial reflexes:** lost plantar reflex in both L.L., lost abdominal reflex (trunkal neuropathy).
- **N.B.:** lost planter reflex may be due to loss on sensation on the sole of the foot, LMNL at SI, weakness in muscles of the big toe or skeletal deformities in big toe).
- ❖ **Back** No deformity, no swelling, no scars
- ❖ **Gait** stamping (may be high steppage)
- ❖ **Other system examination :** search for autonomic neuropathy. See before
- ❖ **Investigation**
 - **For diabetes:** Bl. Sugar level with HBAIC, ECG, RFTs, blood lipid profile (cholesterol, HDL, LDL, TG)
 - **For P.N:** Nerve Conduction velocity.
- ❖ **Treatment :**
 - **For diabetes:** tight control.
 - **For the P.N.:** Tegretol, gabapentin, vitamins, aldose reductase inhibitor (disappointing results).
- ❖ **Diagnosis Diabetic Peripheral Neuropathy**
- ❖ **N.B.s:**
 1. Lost **abdominal reflex** in this case may be due to trunkal neuropathy.
 2. **Abdominal muscles** power can't be examined by resistance because of proximal myopathy → the patient has history of Thyrotoxicosis.
 3. Lost **knee reflex** → not related to high stock level as lost superficial sensation has nothing to do with deep reflexes but is related to lost deep sensation at the level of the knee (evidenced by lost vibration sense at the knee and may be due to amyotrophy due to femoral neuropathy).
 4. No **muscle wasting** → mainly sensory

Myopathy

❖ Character :

1. Systemic degeneration :
 - Bilateral , symmetrical
 - Gradual onset, progressive course
2. Triad of : Weakness + wasting + hypotonia
- Distribution of weakness in myopathy : **هَام هَام هَام**, bilateral, symmetrical :
 - Proximal more than distal
 - Adductors more than abductors
 - Extensors more than flexors
3. No sensory affection, fasciculations or sphincteric disturbances

❖ Signs due to weakness of the proximal muscles :

❖ Affected muscle	❖ Effect
Serratus anterior and lower fibers of trapezius	Winging of the scapulae
Weakness of the extensor muscles of the trunk	Exaggerated lumbar lordosis
Weakness of the abdominal muscles.	Pot-belly abdomen
Weakness of the gluteus medius & minimus (abductors of the hip).	Waddling gait , wide base gait, pelvis is tilting during walking
Weakness of gluteus maximus	Gower's sign Characteristic manner in getting up from the floor (climbing himself)

- N.B.: other causes of waddling gait : hip dislocation & congenital

❖ How to reach diagnosis ?

- Triad of weakness, wasting, hypotonia signifies that our case is muscle disease either: myopathy, myasthenia, myotonia :
- 1) Myasthenia can be excluded by absence of diurnal variation and descending march.
- Test for fatigability to exclude myasthenia : ask patient to :
 - Look up for 1 min. : ptosis will occur

- 2) Myotonia can be excluded by absence of myotonic phenomenon.
- 3) So the case is most probably myopathy.

❖ Causes of myopathy:

- Hereditary (Progressive muscular dystrophy).
- Trauma → history
- Drugs e.g. corticosteroids, Alcohol → History
- Endocrinal: Acromegaly, Cushing, myxedema, thyrotoxicosis → History
- Inflammation → fever, tenderness + skin changes.
- So our case is most probably hereditary:

❖ Hereditary myopathy may be either: pelvic girdle or shoulder girdle, distal type of Gower, ocular type

- Ocular type is excluded by intact ocular muscles
- Distal type of Gower is excluded by examination if proximal muscle affected more than distal
- Then ask the patient when did the weakness start:

- If in LL first:

Pelvic girdle type:

Type	1. Pseudohypertrophy		2. Atrophic Leyden-Mobius type
	Duchenne type	Becker type	
Age of onset	○ 1 st decade	○ 2 nd decade	
Course	○ Malignant (rapidly progressive)	○ Benign (slowly progressive)	
Association	○ Skeletal deformities e.g. Pes cavus, talipes equinus	○ Absent	
	○ ECG changes		

- If start in UL first:

Shoulder girdle type

A. Fascio-scapulo-humeral type	B. Scapulo-humeral type
▪ Landouzy & Dejerine type	▪ Erb type
▪ Bilateral weakness of facial muscles	▪ Facial muscles are free

❖ Examination of muscles of myopathy :

- In myopathy, there is affection of some muscles and sparing of others (selective sparing or selectivity)

❖ Special muscles test for myopathy

I. Affected muscles

❖ Latismus dorsi



- مسنولة عن حركة شد الجرس
- أفق من وراء العيان وخليه يعمل زاوية 90° بذراعه للجانب وخط ايدك تحت كوعه وقول حاول تنزل ذراعيك
- طريقة أخرى : العيان يحط ايدته في وسطه ونطلب منه يكح ونحس ال posterior axillary fold

❖ Serratus anterior



❖ Rhomboids



- مسنولة عن عمل support to scapula
- مش هتتعرف تشوف قوتها بس ممكن تشوف تأثيرها
- قول للعيان يزق الحيطه ويص على ظهره winging of scapula

- العيان يحط ايدته في وسط ويرجع ذراعه للخلف ضد مقاومة ايدك ونحس العضلة

❖ Pectoralis major : sternal head



• العيان يحط ايده في وسطه ويترك ايديه في وسطه جامد ونحس العضلة

❖ Trapezius : lower fibers



• من وراء العيان خليه يعمل زاوية 90° بذراعه للجانب وحط ايديك فوق ذراعه وقوله حول ترفع ذراعك

❖ Abdominal muscles : Beevor's sign: see before

❖ Pectoralis major : clavicular head



• العيان يرفع ذراعه للأمام ويتني ذراعه ويحاول يشبكهم ويترك ايديه في بعض وحس العضلة

II. Spared muscles :

❖ Trapezius : upper fibers



• من وراء العيان حط ايديك على كتافه وقوله ارفع كتافك ... متخلنيش انزلهم

❖ Sternomastoid :



❖ من قدام العيان :

1. كل عضلة لوحدها: (كل عضلة تترك الرقبة الناحية العكسية)
 - حط ايديك على جنب فكه وقوله زق ايدي وحس العضلة الناحية الثانية
 - حط ايديك التاتيه بنفس الطريقة وحس العضلة الأخرى
2. العضلتين مع بعض: (العضلتين مع بعض يزقوا الرقبة لتحت)
 - حط ايديك تحت نقه وقوله زق ايدي وشوفهم وحسهم الاتنين سوا

Myopathy, Becker variety

- ❖ **Personal history**
- ❖ **Complaint:** inability to stand up from sitting position, 20 years duration..
- ❖ **HPI:**
 - The condition started 25 years ago by gradual onset and slowly progressive course of **weakness** associated with **flaccidity** and proximal **wasting**, with no **muscle twitches**. This weakness affected both lower limbs and progressed 2 years later to involve the upper limbs. It affects **proximal** more than distal muscles, **extensor** more than flexor muscles, **adductor** more than abductor muscles. No **diurnal variation**, no **descending march course**, no **fever**, no **trauma**, no history suggesting **cushing**, **acromegaly**, **hypo or hyperthyroidism** with no history of **drug intake**.
 - 5 years later, the weakness became more **severe** and the patient was unable to **stand up from sitting position** at all.
 - No symptoms of **sensory** affection.
 - No symptoms of **sphincteric** affection.
 - No symptoms of **cranial nerves** affection.
 - No symptoms of increased **I.C.T.**
 - No symptoms of **speech** disorders.
 - No symptoms suggesting **pulmonary or systemic congestion** or any other symptoms suggesting **CVS** affection.
 - No symptoms suggesting other **system** affection.
- ❖ **Past history:**
 - No past history of drugs known to cause myopathy (e.g. Corticosteroids, Chloroquine,...).
 - No past history of D.M, HPN, or operations.
- ❖ **Family history:**
 - No similar condition in family.
 - No consanguinity.
 - No common disease in family
- ❖ **General examination:**
 - **Temperature:** 37.2° c.
 - **Bl. Pressure:** 130/70.
 - **Pulse:** regular, 70 beat/minute, average volume, no special character, vessel wall not felt, equal in both sides with intact peripheral pulsation.
 - **Mentality:** The patient is fully conscious, well oriented for time, place and person. Average mood and memory. The patient is co-operative with average intelligence.
- ❖ **Local examination:**
- ❖ **Examination of Speech:** Normal.
- ❖ **Examination of Cranial Nerves:** Normal. (special care to exam of ocular group and facial nerve).
- ❖ **Examination of motor system :**
 - ↳ **Inspection**
 - Bilateral symmetrical Proximal wasting in upper & lower limbs with pseudohypertrophy in the calf muscles.
 - No skeletal deformities, no trophic changes, no involuntary movement.
 - ↳ **Examination of tone**
 - Bilateral symmetrical Hypotonia in both upper and lower limbs. It's proximal more than distal.
 - ↳ **Percussion**
 - No fasciculation or myotonia.
 - ↳ **Examination of muscle power**
 - Bilateral symmetrical Weakness in both upper and lower limbs. It is proximal more than distal, adductors more than abductors, extensors more than flexors.
 - Serratus anterior, Latismus Dorsi, Lower head of pectoralis major, lower fibers of trapezius are affected while Sternomastoid, Upper fibers of trapezius, Upper head of pectoralis major are spared.
 - Power of abdominal muscles → Beevor's sign.
 - ↳ **Coordination**
 - Coordination cannot be examined in both upper and lower limbs because of weakness.

- ❖ **Reflexes**
 - Bilateral areflexia in upper and lower limbs
 - Babinski : plantar flexion on both sides (normal).
 - Abdominal reflex : diminished below the level of the umbilicus.
- ❖ **Sensory**
 - Superficial sensations: intact.
 - Deep sensation: intact.
 - Cortical sensation : intact.
- ❖ **Back** Exaggerated lumbar lordosis.
- ❖ **Gait** waddling gait.
- ❖ **No affection of other system examination**
- ❖ **Investigation** : EMG, Muscle Biopsy, enzymes.
- ❖ **Diagnosis** :

❖ **Muscle disease, Progressive muscular dystrophy, Pseudohypertrophic pelvic girdle type, Becker variety.**

▪ Wasting, Weakness, Hypotonia, Areflexia	✓ So the lesion is LMNL
▪ Not A.H.C: No Fasciculation.	✓ So the case is Muscle disease
▪ Not Peripheral Nerve: No sensory or sphincteric disturbance.	
▪ Not myoneural junction: No diurnal variation, no descending march course.	✓ So the case is myopathy
▪ Not Myotonia: ○ No Mechanical Myotonia, No Voluntary Myotonia ○ Sternomastoid Muscle is spared.	
▪ Not Ocular: ocular muscles are intact.	
▪ Not distal type of Gower: distal muscle are less affected than proximal.	✓ So the case is either pelvic or shoulder girdle
▪ Pelvic Girdle: ○ LL > UL. ○ Beevor's Sign. ○ Diminished abdominal reflex below the level of umbilicus.	✓ So the case is either atrophic or pseudohypertrophic
▪ Pseudo-hypertrophy: calf muscles are hypertrophied but weak.	✓ So the case is either
▪ To make sure ask for EMG and muscle biopsy.	✓ Duchenne or Becker
▪ Becker ○ 3rd Decade. ○ Slowly progressive. ○ No skeletal deformity. ○ No C.V.S affection.	

❖ **Other case scenario :**

1. Wasting, weakness & hypotonia are more in **upper limb**.
2. **Facial** muscles are affected.

→ So the case is shoulder girdle type, **facio-scapulo-humeral** variety (Landouzy and Dejerren's)

Ataxia

❖ **Definition** : incoordination of voluntary motor activity in the absence of motor weakness with or without disequilibrium

❖ **Types** :

A. Cerebellar	B. Sensory	C. Vestibular	D. Mixed
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لو اختبارات ال co-ordination +ve ← تشك أن الحالة ataxia

لازم تتأكد عن طريق اختبارات ال cerebellar ataxia

لازم تعرف المكونات علشان تعرف النوع , هل هي Friedreich ولا Marie ؟

I. **Cerebellar ataxia**: Signs of incoordination : when eye both opened & closed: see before

Causes of cerebellar ataxia

1. Focal	2. Systemic degeneration (herido-familial ataxia)	1. Disseminated e.g. DS
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❖ **Herido-familial ataxia** : lesion must affect at least cerebellum & pyramidal tract

	Marie's ataxia	Friedrich's ataxia
▪ Ataxia	○ Pure cerebellar ataxia	○ Mixed ataxia
▪ Incidence	○ The simplest form ○ Male > female	○ The commonest form ○ Female > male
▪ Family history	○ Negative	○ Positive (autosomal recessive)
▪ Age of onset	○ 2 nd and 3 rd decade of life	○ 1 st decade of life
▪ Pathology	○ Degeneration of 2 areas in spinal cord	○ Degeneration of 4 areas in spinal cord
▪ Cerebellar tract	○ Neocerebellar affection with characters of cerebellar ataxia ○ zigzag gait	○ Archicerebellum affection with Characters of cerebellar ataxia ○ Standing : swaying (trunkal ataxia) ○ Walking : Wide base "drunken" or staggering gait
▪ Δ tract	○ Marked (+ve Babinski sign)	○ Affected (+ve Babinski sign)
▪ PN	○ Not affected	○ Affected; stock & glove hyposthesia
▪ Posterior column	○ Not affected	○ Affected (sensory ataxia, decrease deep sensation; movement, position, vibration)
▪ Speech	○ Staccato speech or Slurred speech	○ Staccato speech or scanning speech
▪ Sensation	○ Preserved both	○ Impaired superficial sensation ○ Impaired deep sensation (sensory ataxia)
▪ Tone	○ Spasticity (marked pyramidal lesion)	○ Flaccidity (due to associated peripheral neuritis, posterior column, cerebellar lesion)
▪ Deep reflexes	○ Increased (pyramidal lesion)	○ Diminished or lost ○ Lost ankle reflex due to peripheral neuropathy ○ Preserved or exaggerated knee reflex due to pyramidal tract lesion
▪ Associated finding	○ Mental impairment ○ Ocular never palsies ○ Extra pyramidal manifestations e.g. fanning of other toes during planter reflex	○ Skeletal deformities (pes cavus + kyhposcoliosis) ○ Congenital heart diseases & ECG changes, cardiomyopathy ○ Peroneal muscle atrophy ○ Abnormal GIT or DM
▪ Treatment	Antioxidants as co-enzyme Q, new groups (mitoquinone, idebenone)	

Friedreich's ataxia

- ❖ **Personal history :....**
- ❖ **Complaint:** staggering gait since he was 4 years old.
- ❖ **HPI:**
 - The condition started since he was 4 years old by gradual onset and progressive course of gait disturbances ,
 - difficulties in speech, kinetic tremors in both hands and feet with unsteadiness during eye closure.
 - 6 months later, the patient experienced weakness associated with flaccidity and falling of hair with brittling of nails with no twitches or wasting. This weakness affected both LLs. it is in distal more than proximal muscles, in flexor more than extensor muscles, in abductor more than adductor muscles.
 - The condition was associated with loss of sensation in both hands and feet and the patient felt as if he walked on cotton.
 - No symptoms suggesting cranial nerve affection.
 - No symptoms suggesting increased I.C.T. or sphincteric disturbances.
 - No symptoms suggesting pulmonary or systemic congestion or any other symptoms suggesting CVS system affection.
 - No symptoms suggesting other system affection.
- ❖ **Past history:**
 - No History of D.M, HTN, drugs or operations.
- ❖ **Family history:**
 - +Ve family history (his sister suffers from the same condition).
 - +Ve consanguinity: 2nd degree.
 - No common disease in family.
- ❖ **General examination**
 - **Temperature:** 37.2° c.
 - **BL Pressure:** 120/80.
 - **Pulse:** regular, 70 beat/minute, average volume, no special character, vessel wall not felt, equal in both sides with absent peripheral pulsation.
 - **Mentality:** The patient is fully conscious, well oriented for time, place and person. Average mood and memory. The patient is co-operative with average intelligence.
 - **Head & neck:**
 - Head nodding,
 - Bilateral horizontal spontaneous monophasic nystagmus.
 - **UL :** intentional kinetic tremors on doing tests of coordination, hypotonia.
 - **LL:** trophic changes , weakness of pyramidal tract distribution, hypotonia, lost ankle with exaggerated knee reflexes.
- ❖ **Local examination:**
- ❖ **Examination of Speech:** Scanning speech.
- ❖ **Examination of Cranial Nerves:** Normal.
- ❖ **Examination of motor system :**
- ↳ **Inspection**
 - loss of hair and brittle nails in both L.L.
 - No muscle wasting, no skeletal deformities, no involuntary movement, no specific posture.
- ↳ **Examination of tone**
 - Bilateral symmetrical hypotonia in both upper and lower limbs.
- ↳ **Perussion :** No fasciculation or myotonia.

❖ Examination of muscle power

- Normal power in both upper limbs.
- Bilateral symmetrical Weakness in both lower limbs. It is in distal more than proximal muscles, in abductor more than adductor muscles, in flexor more than extensor muscles → (weakness of UMNL).
- Abdominal muscles → normal power.

❖ Coordination

- Coordination cannot be examined in both lower limbs because of weakness.
 - In both Upper limbs:
 - intentional kinetic tremors in eye proved by finger to nose test, finger to finger test and finger to doctor's finger test.
 - Intentional kinetic tremors with hypermetria in eye closure proved by finger to nose test and finger to finger test.
- Loss of buttoning and unbuttoning.
- Adiadokokinesia.
- +Ve rebound phenomenon.
- Head nodding
- Scanning speech
- Bilateral, monophasic, horizontal, spontaneous nystagmus (not cerebellar nystagmus).
- No titubation of the trunk

❖ Reflexes

- Deep reflexes:
 - In both upper limbs: normoreflexia.
 - In both lower limbs: lost ankle (P.N) and exaggerated knee (ΔT lesion) with +ve patellar, adductor reflex.
- Superficial reflexes:
 - +Ve Babinski in both L.L.
 - Abdominal reflexes: preserved.

❖ Sensory

- Superficial sensations: below knee and elbow stock and glove anesthesia.
- Deep sensation:
 - Joint sense lost on both sides.
 - Vibration sense lost at level of medial malleolus, ASIS (post column.+ P.N.).
 - Muscle sense lost, nerve sense lost.
 - +Ve Romberg's test.

- Cortical sensation : can't be examined due to loss of superficial sensation.

❖ Back Normal (No deformities, no pigmentation).

❖ Gait staggering gait.

❖ Other system examination :

- Focus on CVS exam as Friedreich's ataxia may be associated with cardiomyopathy

❖ Investigation :

- For the cause: imaging (C.T & MRI).
- For association: ECG, Echo, CXR, Bl. glucose.

❖ Diagnosis : A case of heridofamilial ataxia for D.D, most probably Friedreich's ataxia

Marie's ataxia

❖ **Personal history :....**

❖ **Complaint:** Inability to reach the mouth with spoon properly during eating, 32 years duration.

❖ **HPI:**

- The condition started since he was 32 years ago by symptoms of in-coordination in the form of **kinetic tremors in both hands and feet** as the patient can't reach his mouth properly with the spoon during eating. This tremor was associated with **staggering gait, difficult speech**, with **difficulty to hold urine when he feels the desire**.
- No symptoms suggesting **Motor** affection.
- No symptoms suggesting **Sensory** affection.
- No symptoms suggesting **cranial nerve** affection.
- No symptoms suggesting increased **I.C.T.** or **sphincteric** disturbances.
- No symptoms suggesting other **system** affection.

○ **Past history:**

- No History of D.M, HTN, drugs or operations.

❖ **Family history:**

- No family history of similar conditions.
- No consanguinity.
- No common disease in family.

❖ **General examination**

- **Temperature:** 37.2° c.
- **Bl. Pressure:** 120/70.
- **Pulse:** regular, 70 beat/minute, average volume, no special character, vessel wall not felt, equal in both sides with absent peripheral pulsation.
- **Mentality:** The patient is fully conscious, well oriented for time, place and person. Average mood and memory. The patient is co-operative with average intelligence.
- **Head :** Bilateral biphasic horizontal fixation nystagmus with its direction always towards point of fixation, staccato speech with no head nodding.
- **UL:** intention kinetic tremors on doing tests of coordination, hypotonia.
- **LL:** side to side movement on doing heel to knee test, hypotonia.

❖ **Local examination:**

❖ **Examination of Speech:** Staccato speech.

❖ **Examination of Cranial Nerves:** Bilateral biphasic horizontal fixation nystagmus with its direction always towards point of fixation

❖ **Examination of motor system :**

⌚ **Inspection**

- No muscle wasting, no skeletal deformities, no involuntary movement, no specific posture, no trophic changes.

⌚ **Examination of tone**

- Bilateral symmetrical hypotonia in both upper and lower limbs.

⌚ **Percussion**

- No fasciculation or myotonia.

⌚ **Examination of muscle power**

- Normal power in both upper and lower limbs with normal power in abdominal muscles.

❖ Coordination

○ In Upper limbs:

- intentional kinetic tremors proved by finger to nose test, finger to finger test, finger to doctor's finger test during eye opening and finger to finger test, finger to nose test during eye closure.

○ In Lower limbs:

- There is side to side movement in heel to knee test during eye opening and closure.
- Loss of buttoning and unbuttoning.
- Dysdiadokokinesia.
- +Ve rebound phenomenon.
- Lost heel to toes gait.
- Head nodding, No titubation of trunk.
- Bilateral biphasic horizontal fixation nystagmus with its direction always towards point of fixation.
- Stacatto speech.
- +ve Romberg's test .

❖ Reflexes

- **Deep reflexes:** Bilateral symmetrical normoreflexia in ULs and LLs.
- **Superficial reflexes:** +ve Babiniski i.e dorsiflexion in big toe (pyramidal tract lesion) with fanning of other toes and withdrawal in both L.L. (Extra ΔT lesion).

❖ **Sensory:** All sensations (Superficial, deep, cortical) are intact.

❖ **Back Normal** (No deformities, no pigmentation, no swelling ,no scars).

❖ **Gait** Zigzag gait.

❖ **No affection in other system examination**

❖ **Diagnosis :** A case of heridofamilial ataxia, most probably Marie's ataxia.

Motor neuron disease; MND

- ❖ **Definition** : systemic degenerative disease with unknown cause, of gradual onset & progressive course affecting motor system only with intact sensation, there is degeneration of UMN or LMN or both.
- ❖ **Types** :

A. Anatomical classification			B. Functional classification		
○ Spinal type: spinal cord lesion	○ Bulbar type: brainstem lesion	○ Spino-bulbar type: mixed lesion	○ Pure UMN	○ Pure LMN	○ Mixed type

❖ Clinically :

1. Pure UMN affection: Bilateral pyramidal tract affection :

A. Spinal MND	B. Bulbar MND
○ Lesion in spinal cord	○ Lesion in the brain stem or the cerebral hemisphere
↳ Primary lateral sclerosis	↳ Pseudo bulbar palsy
▪ Degeneration of corticospinal tract, bilateral sign & symptoms of UMN (paralysis, hypertonia, hyperreflexia, no or mild wasting, no trophic changes) resulting in : ➢ Spastic Paraplegia (LL) : if the lesion below cervical region or ➢ Spastic Quadriplegia (4 limbs) : if the lesion above cervical region	▪ Degeneration of corticobulbar tract, results in clinically tetrad of : dysphagia, dysarthria, dysphonia (hoarseness of voice), nasal regurgitation, other clinical picture depend on the site of the lesion: 1. Above medulla: ➢ Spastic Quadriplegia with UMN signs in UL & LL ➢ Pseudo bulbar palsy (Exaggerated palatal, pharyngeal reflexes) 2. Above pons: ➢ As above medulla ➢ Appearance of Jaw reflex 3. Above midbrain: ➢ As above pons ➢ Emotional & mood changes
C. Spinal- bulbar MND: spinal MND & bulbar palsy	

2. Pure LMN affection:

A. Spinal MND	B. Bulbar MND
○ Lesion In AHCs	○ Lesion in Degeneration of cranial nerve nuclei
↳ Progressive muscular atrophy / primary muscular amyotrophy	↳ True bulbar palsy
▪ Degeneration of AHCs with signs of LMN (paralysis, hypotonia, hyporeflexia, wasting) ▪ Commonest site in lower cervical & lumbar region hence it starts early in UL distally (hands → forearm → shoulder) and late in LL ▪ Nerve Affect S1 so no Babinski sign	▪ Clinically tetrad of : dysphagia, dysarthria, dysphonia (hoarseness of voice), nasal regurgitation with : ○ Absent reflexes: palatal, pharyngeal ○ Tongue is wasted showing fasciculations ○ No quadriplegia, no emotional changes
C. Spinal- bulbar MND: spinal MND & bulbar palsy	

3. Mixed type: Combined UMN & LMN affection:

Amyotrophic lateral sclerosis (ALS):

- Amyotrophic = atrophic = LMN
- Lateral sclerosis = Δ tract = UMN
- Combined signs & symptoms of upper & lower motor neuron lesion:

↳ UMN :	↳ LMN:
○ Pyramidal tract lesion	○ AHCs lesion
○ + ve Babinski sign	○ Wasting
○ Weakness of Δ tract distribution	○ fasciculations
→ Tone & deep reflexes according to which one is predominant Δ tract or AHC lesion	

- N.B.: Tonic atrophy : hypertonia & hyper-reflexia + wasting & weakness in mixed type of MND in which Δ tract lesion is predominant
- B. Mixed bulbar palsy (mixed bulbar palsy & similar picture of combined affection)

في حالات ال MND لا تنسى فحص ال Bulbar CN

❖ Differential Diagnosis of MND:

- Other causes which cause Δ tract , AHC lesion e.g. cervical spondylosis , syringomyelia
- لاحظ ان في حالات ال cervical spondylosis , syringomyelia العيان عنده sensory manifestation
- ولكن في حالة ال MND ← NO sensory changes at all

❖ أثناء فحص العيان في العملي هتلاقي كل شين متفق مع كونه MND ولكن هتلاقي 3 اعتراضات على تشخيصك :

1. المرض systemic disease, bilateral symmetrical ولكن في الحالة هتلاقي one side affected more than other
2. المرض بيحي في old age وحالتك هتلاقيها young age
3. المرض fatal & survival rate is about 2 years so it's more dangerous than malignancy هتلاقيها أكثر من سنتين

❖ هذه الاعتراضات يمكن تفسيرها كالاتي :

1. One side affected more than other :
- Patient is e.g. right handed, manual worker (MND is a disease of exhausted muscle)
2. Patient is young age:
- ALS type can affect young age
3. Survival rate > 2years:
- Prognosis depends on :

	Good prognosis	Bad prognosis
Fasciculations	↓↓	↑↑
Bulbar affection	-	+

MND (Motor Neuron Disease)

❖ Personal history :....

❖ **Complaint:** Heaviness of both upper and lower of 25 years duration.

❖ HPI:

- The condition started 25 years ago by gradual onset of **weakness** associated with stiffness, **marked distal asymmetrical wasting** in both UL and LL and **twitches**. Both weakness and wasting started in both ULs but they are noticeably more on the Rt. Side. Then, they progressed one year later, to affect both LLs being more on Lt. side. Weakness was **distal** more than proximal, in **abductor** more than adductor muscles, in **extensor** more than flexor muscles in U.L., while in L.L., it is more in **flexors** than extensor muscles with no involuntary movements.
- No symptoms of **sensory** affection.
- No symptoms of **Bulbar or Cranial Nerve** affection.
- No symptoms of **sphincteric** affection.
- No symptoms of increased **L.C.T.**
- No symptoms of **speech** disorders.
- No symptoms suggesting **other system** affection.

❖ Past history:

- No past history of DM, HTN, fever, Bilharziasis, drugs or operations.

❖ Family history:

- No family history of similar conditions.
- No consanguinity.
- No common disease in family.

❖ General examination

- **Temperature:** 37.2° c.
- **Bl. Pressure:** 120/70.
- **Pulse:** regular, 70 beat/minute, average volume, no special character, vessel wall not felt, equal in both sides with intact peripheral pulsation.
- **Mentality:** The patient is fully conscious, well oriented for time, place and person. Average mood and memory. The patient is co-operative with average intelligence.

❖ Local examination:

❖ **Examination of Speech:** Normal.

❖ **Examination of Cranial Nerves:**

○ **Bulbar Cr. Nerves** → free.

○ **Other Cranial Nerves** → free.

❖ **Examination of motor system :**

❖ Inspection

○ **Bilateral asymmetrical distal wasting** in ULs and LLs. It's more in right UL and left LL.

○ **Bilateral Pes cavus**, no trophic changes, no involuntary movement, no specific posture.

❖ Examination of tone

○ **Bilateral asymmetrical Hypertonia** in upper limbs and lower limbs. It's more in right UL and left LL.

❖ Percussion

○ **few fasciculation** scattered in right UL and left LL with No myotonia.

❧ Examination of muscle power

- Bilateral asymmetrical Weakness in upper limbs and lower limbs. It's more in right UL and left LL. It is distal more than proximal, in abductor more than adductor muscles, in extensor more than flexor muscles in UL while in LL, It's more in flexors than extensor muscles.

❧ Coordination

- Coordination cannot be examined on both upper and lower limbs because of weakness.

❧ Reflexes

- **Deep reflexes:** Bilateral asymmetrical hyper-reflexia in upper limbs and lower limbs. It's more in right UL and left LL with +Ve pathological reflexes
- **Superficial reflexes:** +Ve Babiniski on both sides. Abdominal Reflex → Lost.

- ❧ **Sensory:** All sensations (Superficial, deep, cortical) are intact.

- ❧ **Back Normal** (No deformities, no pigmentation, no swelling, no scars).

- ❧ **Gait :** patient can't walk

- ❧ **No affection in other system examination :**

- ❧ **Diagnosis :** A case of motor neuron disease, most probably Amyotrophic Lateral Sclerosis .

1. Why MND?

- Purely motor with no sensory or sphincteric manifestations
- Unknown etiology.
- It affects motor neuron only inside CNS.

2. Why spinal type?

- Bulbar and spino-bulbar types are excluded because bulbar cranial nerves are free.

3. Why ALS?

- Because the patient has wasting and fasciculation which are features of LMNL and at the same time, It has weakness of pyramidal tract distribution with +ve planter reflex which are features of UMNL.

- ❧ On the light of these confusing data, the case is either:

1- ALS.

2- Cervical spondylosis.

3- Cervical syringomyelia.

→ But because the patient's sensations are intact, It's most probably ALS.

4. Criticism to the diagnosis :

- **Asymmetrical** → may be attributed to the patient's life style as he is manual worker using his right UL and left LL and MND is a disease of exhausted muscles.

- **Long survival while MND is of short duration** → prognostic criteria are:

- Bulbar affection.

- Fasciculation.

→ Our patient has few fasciculation with no bulbar affection which account for good prognosis and prolonged survival.

- **Early onset of the disease while MND is a disease of old age** → ALS can affect young age.

Parkinsonism

Commonest causes of Parkinsonism :

	A. Paralysis agitans	B. Atherosclerosis	C. Post encephalitic
▪ Age	40-60 years	Over 60	Any
▪ Onset	Gradual	Gradual	Any
▪ Course	Progressive	Remission & exacerbation	Acute
▪ Tremors & rigidity	T > R	R > T	Regressive or stationary
▪ Associations	None	- Amnesia - DM, HTN - Ischemic heart disease - Ischemia of ΔT ; pyramidal signs	- Obesity - Diabetes insipidus - Pyramidal signs - Sialorrhea - Greasy face - Oculogyric crisis

❖ **Complaint** : Shaking movements.

❖ **History** : Involuntary movements : أهم حاجة وصف ال

→ Character:

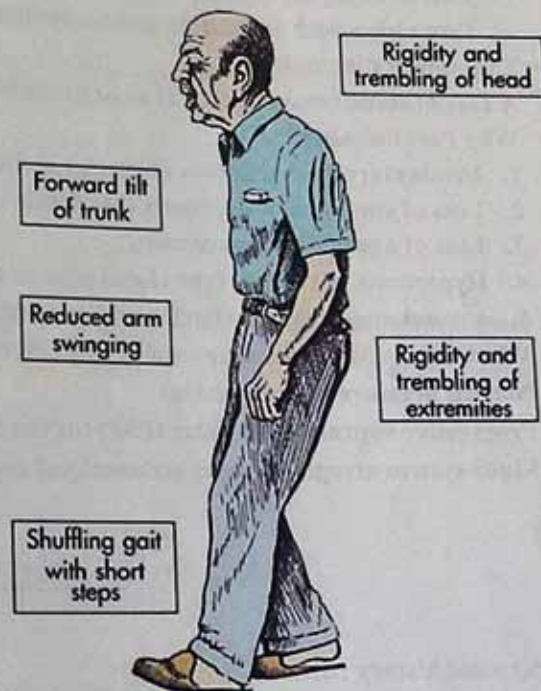
1. Static.
2. Regular.
3. ↑ by emotional stress, anxiety & fatigue قوله قوم امشي هتزيد
4. ↓ by active voluntary movement, sleep. قوله سلم عليا هتقل
5. Form:
 - A. U.L:
 - Fingers: flexion & extension of MPJ (Pill rolling).
 - Wrist: flexion & extension (+ supination, pronation).
 - Elbow: flexion, extension (Severe cases).
 - B. L.L:
 - Toes: flexion & extension.
 - Ankle : flexion & extension.
 - C. Neck:
 - Flexion & extension (Head Nodding).
 - Rotatory movements (rare).
 - D. Head:
 - Mandible: rhythmic opening & closure (rare).
 - Lip tremors.
 - Tongue طلع لسانك وابعدنه عن سناتك

→ Distribution:

1. U.L > L.L
2. Unilateral then bilateral (Asymmetrical).
3. Distal.
4. Present in Head & Neck.
5. Trunk (very rare).

❖ EXAMINATION

1. General:
 - Bl. Pr. → ↑ in atherosclerosis.
 - Facial expression (Mask Face)
2. Mentality → Depression.
3. Speech → monotonous speech (Slow, expressionless).
4. Motor :
 - A. Muscle state:
 - Posture: Generalized flexion attitude (Gorilla-like للإنسانية ماتقولش).
 - Involuntary movements: see before



B. Muscle tone: Hypertonia

- Lead pipe: uniform resistance
- cog wheel: resistance is intermittent & interrupted by tremors
- N.B. Rigidity causes → Slow Shuffling gait & Micrographia.

C. Muscle power:

- Normal power → No weakness

طبيب لو اتسألت ليه اسمه shaking palsy وليه فيه نوع اسمه paralysis agitans قول أسماء خاطئة حيث أن ال rigidity العنيفة هي اللي بتدي إحاء أن المريض يعاني من paralysis

D. Reflexes:

- Hyporeflexia or areflexia
- Hyper-reflexia نادراً If the case is atherosclerotic, pyramidal tract may be also ischemic → UMNL → Hyper-reflexia.

5. Sensory System: Normal sensation.

6. Gait:

- Slow, short steppage → shuffling.
- Loss of swinging of arms.
- Propulsion with festinating gait (sometimes).

❖ Example of diagnosis :

- A case of static tremors for D.D most probably Parkinsonism

❖ Why Parkinsonism?

1. Involuntary movements → character & Distribution ونقولهم
2. Loss of emotional movements (Mask face).
3. Loss of associative movements.
4. Hypertonia of rigidity type (Lead pipe or cog wheel).
5. ± Autonomic changes (Parkinsonism Plus): sialorrhea, greasy face,

❖ What is the differential diagnosis of extra-pyramidal conditions?

- Normal pressure hydrocephalus
- Progressive supranuclear palsy (PSP) (or the Steele-Richardson-Olszewski syndrome)
- Multi-system atrophy causing striatonigral degeneration

Parkinsonism

❖ Personal history :....

- ❖ **Complaint:** Shaking movement of both upper & lower limbs since 1967.

❖ HPI:

- The condition started since 1967 with regular **shaking movement** at rest increase with **emotion** & decrease with **sleep** affect both upper & lower limbs being much more on right side **distal** more than proximal. No similar shaking movement neither in head, neck nor trunk. Associated with **stiffness** in upper & lower limbs being much more in right side leading to **difficulty** in starting movement & **shuffling** gait.

- Not associated with neither **fever**, **trauma**, **vomiting**, **coma** nor **convulsions**.
- No symptoms of **cranial nerve** affection.
- No symptoms of **speech** affection.
- No symptoms of **sphincteric** affection.
- No symptoms of **increased I.C.T.**
- No symptoms suggesting **other system** affection.

❖ Past history:

- No past history of DM, HTN, drugs or operations.

❖ Family history:

- No family history of similar conditions.
- No consanguinity, No common disease in family.

❖ General examination

- Temp.: 37.2° c.
- Bl. pressure: 220/100.
- Pulse: regular, 70 beat/min, average volume, no special ch.ch, vessel wall not felt, equal in both sides
- Mentality: the patient is fully conscious well oriented for timeplace & person, average mood (may be euphoric) & memory. The patient is co-operative with average intelligence.
- Head & Neck: No mask face.

❖ Local examination:

- ❖ Examination of Speech: Normal.
- ❖ Examination of Cranial Nerves: Myerson's sign
- ❖ Examination of motor system :

❖ Inspection

- Generalized muscle wasting (sunken eye, triceps skin fold, temporalis)
- Involuntary movements:

→ U.L:

- Right: asymmetrical flexion & extension of fingers of Rt. hand (pill rolling) movement,
- Left: Normal.

→ L.L:

- Right: flexion & extension of toes & ankle,
- Left: side to side movement at the ankle.

- Tongue tremors, No Nodding, No lip tremors

❖ Examination of tone

- Right upper & lower limbs: marked rigidity of cog wheel ch.ch affecting both flexor & extensor but more in flexors.
- Left upper & lower limbs: very mild rigidity in U.L.

❖ Percussion

- No fasciculation nor Myotonia.

❖ Examination of muscle power

- U.L: Bilateral asymmetrical weakness of pyramidal tract distribution affecting right > left.
- L.L: Bilateral asymmetrical weakness affecting distal > proximal more in right.

❖ Coordination

- Coordination cannot be examined on both upper and lower limbs because of weakness.

❖ Reflexes

- Left U.L & L.L: Normoreflexia.
- Right U.L & L.L: Exaggerated response especially biceps, triceps, brachioradialis, knee,
- Lost planter reflexes on right side & diminished planter on left.
- Pathological reflexes: Positive (patellar & adductor).

- ❖ Sensory: All sensations (Superficial, deep, cortical) are intact.

- ❖ Back Normal (No deformities, no pigmentation, no swelling, no scars).

- ❖ Gait : Slow, shuffling, short steppage, loss of swinging of arms.

- ❖ No affection in other system examination :

- ❖ Diagnosis : A case of static tremors for D.D most probably Parkinsonism

Disseminated sclerosis Multiple sclerosis

❖ Definition :

- Disseminated de-myelinating disease of unknown cause (may be autoimmune, viral infection)

❖ Pathology : Demyelination, sclerosis.

❖ Course : Remission, exacerbation.

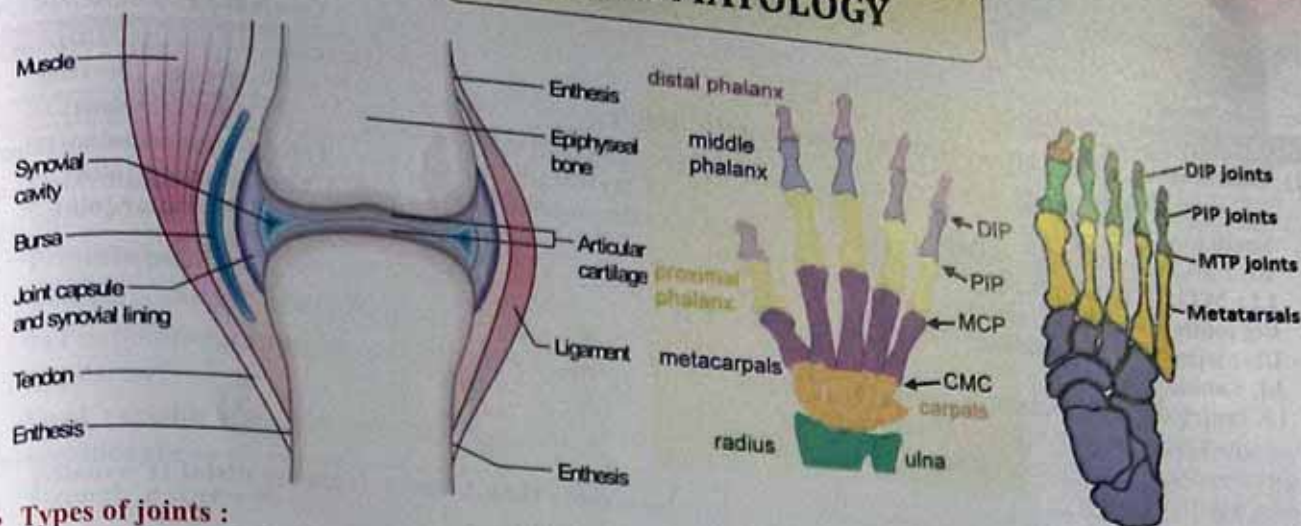
❖ Precipitating factors:

○ Fever, infection.	○ Physical fatigue.	○ Emotional stress.
○ Operations.	○ Pregnancy.	

❖ Signs & symptoms :

1. Mentality changes.
 - Euphoria or Depression or Emotional lability (uncontrolled burst of crying or laughing)
 2. Speech disturbances; Dysarthria:
 - Slurred or Staccato or Scanning.
 3. Cranial nerve involvement specially:
 - A. The optic nerve resulting in:
 - Diminution or loss of vision due to optic neuritis or primary optic atrophy.
 - Temporal pallor of optic disc on fundus examination.
 - Visual field defects and disturbances of colour vision
 - B. The ocular nerves (3,4,6) resulting in:
 - Diplopia which is a common early symptom.
 - Ophthalmoplegia
 - Ophthalmoplegia-internuclearis (pathognomonic)
 - Due to lesion of the medial longitudinal bundle (MLB) which carries the fibres of the conjugate eye movements.
 - On looking to one side there is:
 - ↳ Nystagmus in the abducting eye.
 - ↳ Weakness or paralysis of the adducting eye.
 - C. Trigeminal neuralgia : early symptom
 - D. The facial nerve resulting in:
 - Facial palsy of U.M.N.L. (common) or L.M.N.L. (very rare).
 - Hemifacial spasm (pathognomonic)
 - E. The cochleo-vestibular nerve : vertigo
 - F. Glossopharyngeal & vagus : bulbar palsy : rare
 4. Motor System affection:
 - Form : monoparesis, hemiparesis, paraparesis, quadriparesis or pseudo-bulbar palsy.
 - This is associated with signs of U.M.N.L. i.e. hypertonia, hyperreflexia, +ve Babinski sign and early loss of the abdominal reflexes.
 5. Sensory System affection:
 - Transient numbness and paraesthesias followed by superficial and/or deep sensory loss but no peripheral neuropathy is present
 - +ve L'hermite's sign may be present: on flexion of the head there is sudden electric like sensation radiating to the back and limbs, it is due to posterior column involvement in the cervical region.
 6. Cerebellar affection:
 - Cerebellar ataxia is a common presentation; it is associated with nystagmus, staccato speech, intention kinetic tremors and gait disturbances.
 7. Autonomic disturbances:
 - Precipitancy, hesitancy of autonomic bladder.
 - Impotence.
- ❖ N.B.: early findings in DS
- Optic neuritis, Lost abdominal reflexes & Sphincteric disturbances and impotence

RHEUMATOLOGY



Types of joints :

1. Peripheral joint	2. Axial joints	3. Specific joints
• Big : ○ UL: wrist, elbow, shoulder ○ LL: ankle, knee, hip • Small : the rest	• Cervical , thoracic, lumbar	○ Temporomandibular joint (TMJ) ○ Sacroiliac joint (SIJ)

Classification of joints :

According to number of joints:	According to onset & duration:	According to etiology:
• Monoarthritis : 1 joint • Oligoarthritis : 2-4 joints • Polyarthritis : > 4 joints	• Acute : lasting less than 6 weeks • Chronic : lasting more than 6 weeks	• Degenerative (Mechanical) : ○ Osteoarthritis ○ Spondylosis • Inflammatory : ○ Rheumatoid arthritis (RA) ○ Systemic lupus erythematosis (SLE) ○ Ankylosing spondylitis (AS) ○ Idiopathic juvenile arthritis (IJA)

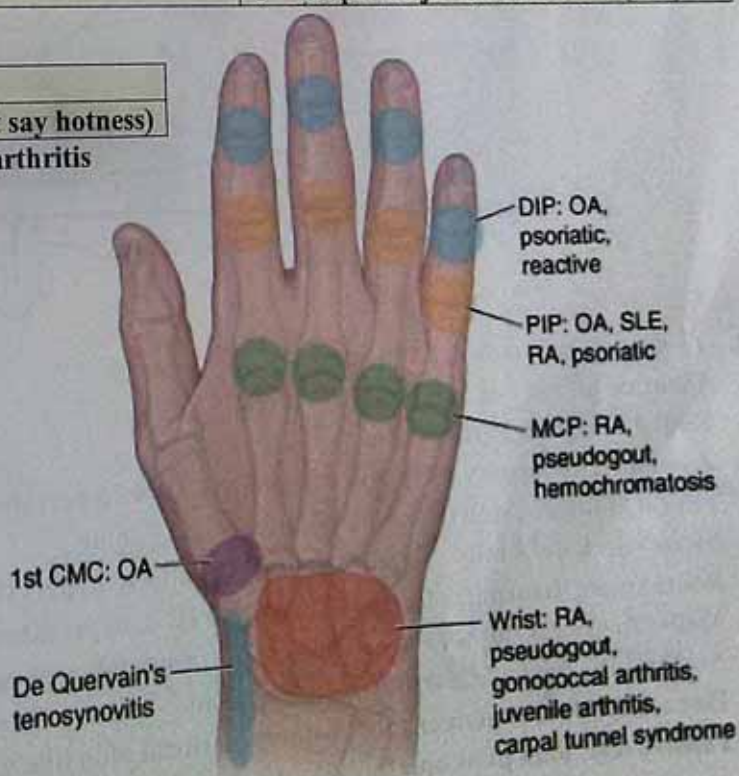
Criteria of the activity of the disease clinically:

1. Swelling	2. Tenderness
3. Erythema (don't say redness)	4. Warm (don't say hotness)

- N.B.: redness & hotness are reserved for septic arthritis

Arthritis of small joints of the hand :

○ DIP: Distal Interphalangeal joint
○ PIP: Proximal Interphalangeal joint
○ MCP: Metacarpophalangeal joint
○ 1 st CMC: 1 st carpometacarpal joint



❖ Rheumatoid arthritis = RA :

❖ Type of patient :

- Sex : female > male; 3 : 1
- Age between 30 - 40 years

❖ Articular manifestations :

1) Distribution : polyarthritis , bilateral, symmetrical affecting :

A. Peripheral joints:	B. Axial joints :	C. Special joints:
1. Small joints : ○ UL : MCP & PIP (Spares DIP) ○ LL: MTP, PIP (Spares DIP) 2. Big joints (after small joints): ○ UL : wrist , elbow, shoulder ○ LL : ankle, knee, hip → i.e. centripetal distribution i.e. polyarthritis starts at small joints (MCP & PIP) then spreads proximally to involve large joints (wrist, Knee, hip, axial)	○ Cervical joints e.g. atlanto-axial joints	○ Temporomandibular joint ○ Crico-artenoid joint ○ Sternoclavicular joint

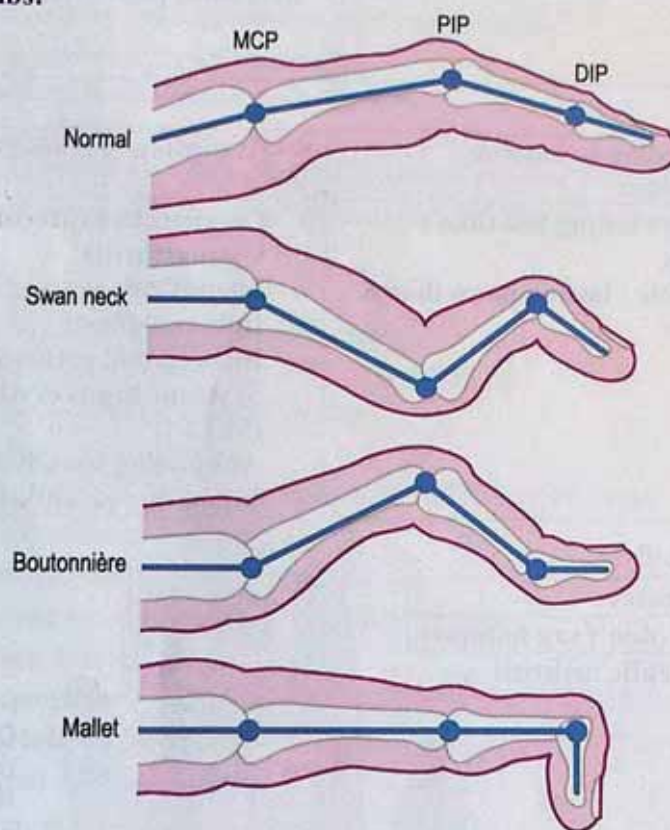
2) Character : Severe Arthritis of joints with morning stiffness more than 1 hours (sparing distal IP usually)

3) Inflammatory cardinal signs : erythema , warmth , swelling & tenderness

4) Deformity : erosive arthritis

A. SMALL joints :

✶ In Upper limbs:



1. Z-shaped deformity of the hand:

- Ulnar deviation : at MP joint (NOT WRIST)
- Radial deviation at the wrist

2. Z-shaped deformity of thumb : flexion at MP & hyperextension of proximal IP

3. Fusiform finger : due to swelling of proximal IP joints

4. Swan - neck deformity : flexion of distal IP joint & hyperextension of proximal IP

5. Boutonniere deformity: flexion of proximal IP & hyperextension of distal IP .

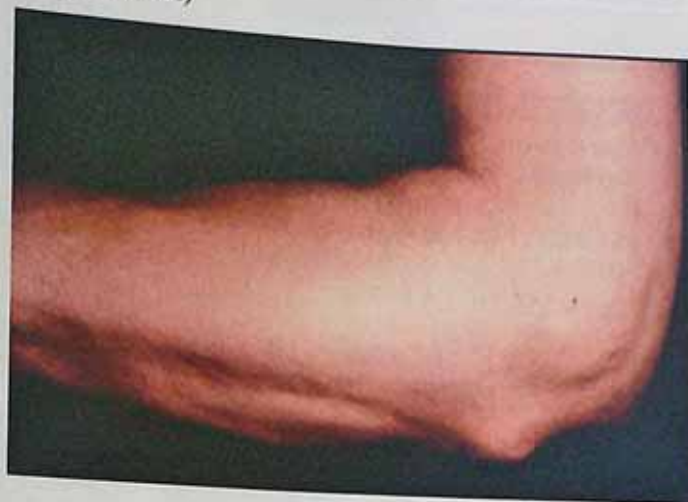
6. Mallet finger : injury to the extensor digitorum tendon → end of a finger is flexed towards the palm

7. Non-specific flexion deformity e.g. claw hand

8. Piano-Key Sign: examiner pushes down on distal ulna like a piano key

- Positive test: ulna pops up after release of the downward pressure

- In lower limbs:
 - Big toe : hallux valgus
 - Small toes : hammer toes (flexion deformity)
- B. **BIG JOINTS** : mainly flexion deformities , genu valgum and genu varum
- ❖ **Extra-articular manifestations:**
 1. Systemic: usually mild, see later with systemic examination of the case
 2. SC nodules :
 - A. Pathology:
 - Central fibrinoid degeneration (necrosis) with inflammatory cells covered by fibrous capsule.
 - B. Site:
 - Pressure points e.g. elbow & knee
 - Extensors surface of the limbs
 - In relation to tendons (tendo-Achilles, extensor tendon of hands)
 - C. Character:
 - Oval, variable size firm, painless, mobile.
 - D. Significance :
 - Strongly positive rheumatoid factor
 - Prognostic : bad prognosis
 - E. Differential diagnosis :
 - SC nodules of rheumatic fever
 - Heberden's & Bouchard nodes of OA
 - Tophi of gout
 - F. Course:
 - Transient, disappear
 - G. Complications
 - Ulceration & infection
 - Rupture tendon : finger drop, foot drop
 - Trigger finger



❖ Type of Patient :

- ✓ Sex: females more than males; 10 : 1
- ✓ Age : 2nd & 3rd decade

❖ Articular manifestations :

- Distribution : like RA
- Character : mild
 - Mild arthralgia (rarely arthritis), myalgia
 - NO deformities

❖ Extra-articular manifestations:

- Usually severe (kidney)
- NO subcutaneous nodules



❖ Systemic Lupus Erythematosus (SLE)

❖ Rheumatic fever:	❖ Gout
• Polyarticular, fleeting, affecting big joints • Associated with other major or minor criteria of Rheumatic fever	• Starts at MP of big toe • May affect distal IP of hand & feet • Acute inflammation + raised serum uric acid • Gouty tophi : ○ Hard, tender, if squeezed → chalky material (urate crystals) ○ Site : dorsum of hand, ear pinna

What are 3 principal crystals associated with joint inflammation?

- Urate : gout
- Calcium pyrophosphate : pseudogout
- Hydroxyapatite : pseudo-pseudogout

❖ Osteoarthritis = Osteoarthritis = OA

- Affecting weight bearing joint in limb or vertebrae
- Pain is only related to movement + coarse crepitus at joint occur
- Osteophytes (hard nodes) in :
 - Distal IP (Heberden nodes) &
 - Proximal IP (Bouchard nodes) in juvenile type



Heberden

Bouchard

❖ Type of Patient :

- ✓ Sex: females more than males
- ✓ Age : mainly elderly

❖ Types of osteoarthritis :

- Primary: e.g. hand : nodal generalized osteoarthritis with Osteophytes (hard nodes) in distal IP (Heberden nodes) & proximal IP (Bouchard nodes) in juvenile type
- Secondary : trauma , inflammation, acromegaly , etc...

Extra-articular manifestations: none

Articular manifestations :

A. Distribution : localized or generalized :

- Peripheral joints :
 - Big: e.g. knee & hip
 - Small :
 - Hands : DIP & PIP, but spares MCP joints
 - Feet: DIP & PIP, but spares MTP joints
- Axial joints : cervical & lumbar

B. Character :

- Crepitus
- Osteophytes (Nodules)
- Minimal deformities

• Which arthritis can affect DIP?

○ Osteoarthritis

○ Psoriasis

○ Juvenile arthritis

○ Gout

❖ Ankylosing spondylitis

❖ Type of Patient :

- ✓ Sex: males more than females; 3 : 1
- ✓ Age : 2nd & 3rd decade (onset over 45)

Articular manifestations :

A. Distribution : polyarthritis

- Axial joint : cervical, thoracic & lumbar
- Special joint : sacroiliac joint
- Peripheral joint : Asymmetrical specially in LL

→ Centrifugal distribution i.e. polyarthritis starts at large joints (axial, hip, Knee, ankle, shoulder, elbow , wrist) then spreads distally to involve peripheral small joints (MCP & PIP)

B. Character :

- Back pain with limitation of movements (Morning stiffness)
- Deformities e.g. kyphosis

Extra-articular manifestations:

- Usually mild
- Specially Iris & aortic Incompetence (AR)



❖ How to differentiate between articular & non-articular arthritis?

Articular	Non-articular
○ Tender joint. ○ Disturbed active & passive movement. ○ Deformity	○ Tenderness outside joint lines. ○ Passive movement is better than active. ○ No deformity. (As bursitis & tendonitis)

❖ How to differentiate between inflammatory & mechanical arthritis?

Inflammatory	Mechanical
○ Increased by rest. ○ Decreased by activity. ○ More in morning.	○ Increased by activity. ○ Decreased by rest. ○ More at night.

❖ Personal History:

RHEUMATOLOGY HISTORY

1. Name: الإسم الثلاثي	2. Age: ○ Child : still's disease & rheumatic fever ○ Middle age : gout, rheumatoid, SLE ○ Old age : osteoarthritis	3. Sex : ○ Female : rheumatoid, SLE ○ Male : ankylosing spondylitis & Reiter's syndrome
4. Occupation: ○ May predispose to osteoarthritis	5. Marriage 6. Menstrual history if female.	7. Residence & address
8. Habits of medical importance		9. Handedness.

❖ Complaint + duration: ؟

❖ History of present illness = HPI : Pain in both hands

1) Pain هل مفاصلك فيها ألم ؟

1. Onset: الموضوع بدأ معاك بالتدريج ولا مرة واحدة ؟
- Gradual : rheumatoid arthritis, SLE
 - Acute : gout & rheumatic fever
2. Course: يزيد ولا يقل ولا يبروح ويجي ؟
- Progressive : osteoarthritis
 - Regressive : Rheumatic fever
 - Remittent: rheumatoid & chronic gout

3. Duration : لمدة أدي ؟

- Long: rheumatoid arthritis, SLE
- Short : acute gout & rheumatic fever
- 4. Site : spine lesion may cause radicular pain
- 5. Type : morning stiffness & duration
- In Rheumatoid Arthritis more than 1 hour

هل مفاصلك مخشبة (ناشفة أو مكلبشة) عليكي الصبح ولمدة أدي ؟ وبتطري أو بتفك بعد أدي ؟

• N.B.: morning stiffness may improve by treatment so history of drug intake is important.

6. Precipitating factors: في حاجة بتزود أو تقلل الألم ؟

- Diuretics & diet: precipitate gout
- Throat infection : precipitate rheumatic fever
- 7. Aggravating & relieving factors : يزيد بالراحة ولا بالمجهود

○ Inflammatory arthritis (Rh.arthritis) : ↑ by rest & ↓ by activity

○ Mechanical arthritis (osteoarthritis) : ↓ by rest & ↑ by activity

8. Diurnal variation : الصبح أكثر ولا على نهاية اليوم ؟

○ Inflammatory arthritis : ↑ in morning & ↓ by end of day

○ Mechanical arthritis : ↓ in morning & ↑ by end of day

9. Functional disability : ability to work

○ لسه بتقدري تعملي شغل البيت ولا لا ؟

2) Swellings

- هل مفاصلك ورمت ؟ أي مفصل ؟ هل الورم مستمر ولا يبروح ويجي ؟
- Episodic arthritis (signs of inflammation) : describe frequency & duration
- هل حصل اي التهابات في المفصل قبل كده واحمرت ووجعتك ؟
- SC nodules (extra-articular manifestation)
- هل في كلاكع صغيرة ظهرت لكي قبل كده ؟

3) Deformities هل حصل تشوهات ، في أي مفصل ؟

❖ Pattern of affection :

- هل واخده مفصل واحد ولا أكثر من مفصل ؟
- Monoarticular : suppurative arthritis & hemarthrosis, gout (recurrent in big toe)
- Polyarticular : rheumatoid arthritis, SLE
- Oligoarticular : osteoarthritis
- Fleeting : rheumatic arthritis

4) Associated non articular symptoms = Systemic manifestations = Symptoms of other systems :

❖ هل مع التعب ده في أعراض تانية ؟

❖ Multisystem complaints : Rh.A, SLE :

1. Weight loss, low grade fever
2. Ocular manifestations
3. Cardiac symptoms , chest symptoms , GIT symptoms , renal symptoms (nephrotic syndrome)
4. Anemia symptoms
5. Skin : SLE , progressive systemic sclerosis:
- In SLE : In addition, ask for Mucocutaneous changes "DLE":
• Discoid lupus : maculopapular rash in butterfly area.
• Photo sensitivity وشك بيزنهر لما بتقف في الشمس
• Others : hyperpigmentation, vitilligo, alopecia, Raynaud's phenomenon, finger ulcers
6. Abdominal pain & fever: Familial Mediterranean Fever
7. Bleeding PR : enteropathic colitis
8. Bloody diarrhea in SLE
9. Neurologic : Charcot's joint
10. Bleeding tendency : hemarthrosis, Henoch- Shoenlein purpura

5) Investigations & treatments:

- Hospitalization, medical treatment, physiotherapy & surgery

A. Past history as usual but focus on :

- Trauma: hemarthrosis, osteoarthritis
- Raynaud's phenomenon (Pallor, Cyanois, Rubor) : progressive systemic sclerosis
- Psoriasis : in psoriatic arthritis
- Treatment history : previous treatment & its efficacy

B. Family history as usual & asked for:

- Osteoarthritis
- Rheumatoid arthritis
- Gout
- Familial Mediterranean Fever (FMF)

General Examination Of Rheumatologic Case

❖ Examination as general examination but stress on the following:

I. **Vital signs** : Pulse : vasculitis → absent peripheral pulsations

II. Other systems except that of local exam :

Rheumatoid arthritis	Systemic lupus erythematosus
❖ Criteria for diagnosis	
• At least 4 of the following 7 criteria : 1. Morning stiffness more than 1 hour 2. Arthritis of 3 or more joints 3. Arthritis of hand joints 4. Symmetrical arthritis ○ All of the above must be present for at least 6 weeks 5. SC rheumatoid nodules 6. Characteristic X-ray findings 7. Positive rheumatoid factor	• At least 4 or more of the following : 1. Malar or butterfly rash 2. Discoid rash 3. Photosensitivity 4. Painless oral ulcer 5. Peripheral joints arthritis : 2 or more peripheral joints 6. Pleurisy or pericarditis 7. Proteinuria 8. Psychosis or seizures 9. Pancytopenia 10. Positive Antinuclear antibody 11. Positive anti-double stranded DNA antibody or anti smith antibody
❖ General	
○ Fever, fatigue , weight loss ○ Cushingoid features secondary to cortisone therapy	
○ Morning stiffness lasting for 1 hour or more (history)	
❖ Skin	
• Psoriatic arthropathy "seronegative" affect distal IP joints (arthritis mutilans): Psoriasis of skin extend to nail • SC rheumatoid nodules • Palmer erythema • Pallor due to anemia	• Malar rash : erythema over the cheeks • Butterfly rash : erythema on the cheeks & nose • Discoid rash : ○ Erythema , scar formation, scaling , skin pigmentation & telangiectasia occur over the face & neck. • Photosensitivity: Skin rash as a result of unusual reaction to sunlight • Alopecia , vitiligo • Vasculitis : ○ Purpura & leg ulcer ○ Raynaud's phenomenon

1. Conjunctivitis, scleritis with repeated attack can cause scleromalacia (Blue sclera)
2. Ankylosing spondylitis: anterior uveitis
3. Sjogren's syndrome:
 - Arthritis
 - Dry eye (xerophthalmia) = keratoconjunctivitis sicca (Schirmer's test)
 - Dry mouth (xerostomia), Dry skin, Dry vagina
 - Lymphadenopathy: there is increased incidence
 - Renal tubular acidosis & Raynaud's phenomenon
 - Associated auto immune diseases e.g. myasthenia gravis, autoimmune hepatitis, thyroid diseases
4. Reiter's syndrome: sero -ve arthritis:
 - Arthritis, Conjunctivitis, Urethritis
5. Bechet's syndrome: sero -ve arthritis
 - Arthritis, Iritis
 - Oral & genital ulceration
 - Occlusive vasculitis (IVC thrombosis)

❖ Eye

- Conjunctivitis, scleritis, anterior uveitis

❖ Cardiac

- Pericarditis, myocarditis, effusion, Systemic hypertension, ischemic heart disease
- Rheumatoid arthritis: AR due to vasculitis, not due to endocarditis
- Ankylosing spondylitis: AR
- Libman endocarditis (non infective endocarditis) = verrucous endocarditis: aortic & mitral regurge

❖ Chest

- Pleural effusion, pleurisy (rub), interstitial fibrosis, Pulmonary hypertension
- Caplan's syndrome (RA + pneumoconiosis + Rheumatoid nodules in x ray lung)
 - Pulmonary infarction (Shrinking lung syndrome)
 - Acute respiratory distress syndrome

❖ CNS

- Peripheral Neuropathy: peripheral neuritis & carpal tunnel syndrome **الخصب** (see later in endocrinology)
- Myopathy
- Cerebral vasculitis: stroke, Chorea, Depression, Epilepsy
- Atlanto-axial subluxation: spinal cord compression → paraplegia
- Wasting of the small muscles of the hand
- Transverse myelitis

❖ GIT

1. Felty's syndrome:
 - Arthritis
 - Splenomegaly with hypersplenism
 - Pancytopenia, Lymphadenopathy
2. Still's disease:
 - Fever, Skin rash, Splenomegaly
 - Rheumatoid Factor: usually negative
 - Late arthropathy so misdiagnosed as hematological disorder.
3. Hepatosplenomegaly: amyloidosis if long standing
4. Palpate iliac fossa for Crohn's disease & ulcerative colitis (colitic arthropathy - seronegative)
5. Urethra: urethritis in Reiter's syndrome "seronegative"
- Lymphadenopathy
- HepatoSplenomegaly
- Painless oral ulcers

❖ Renal

- lupus nephritis: acute or chronic renal failure

- Lower limb edema due to:
 - Nephrotic syndrome to amyloidosis or drug induced GN
 - Pulmonary hypertension or IPF: cor pulmonale
 - Myocarditis: HF

Local Examination Of Rheumatologic Case

1) Inspection

2) Palpation

3) Movements (Motion)

1) Inspection:

1. Deformity:

- A. In UL & LL: see before Boutonniere, Swan neck, Hallux valgus ... etc.
- B. Back : بصر مرة من قدام ومرة من الجنب ومرة من وراء
- From the side, for cervical, thoracic & lumbar curves e.g. thoracic Kyphosis or cervical & lumbar lordosis
 - From behind , for scoliosis; an imaginary line drawn from T1 should fall through the gluteal cleft
 - Kyphosis (DD postural & structural) :
 - Postural kyphosis disappears by axillary suspension of the patient or putting him in prone position i.e. correctable

2. **Distribution:** symmetrical peripheral polyarthropathy centripetal extension in rheumatoid arthritis

3. Description:

A. Skin :

- Erythema (acute arthritis), swelling
- Palmer erythema
- Scar for operation (synovectomy) or arthroscopy or trauma
- Trophic changes, scaly skin lesions & nail pitting in Psoriasis

B. Joint contour : for displacement (Subluxation)

- The most common displacement is at MCP
- The most dangerous displacement is atlanto-axial joint

C. Swellings :

A. SC nodules :	B. Fluid : Effusion :	C. Bone swelling :
▪ Sites : A. Pressure points e.g. elbow & knee B. Extensors surface of the limbs C. In relation to tendons (tendo-Achilles, extensor tendon of hands)	▪ Sites : A. IPJ : fusiform finger B. MCP J: obliteration of spaces between knuckles C. Wrist: diffuse swelling D. Knee : swelling around patella	▪ In osteoarthritis ▪ Sites : A. Distal IP (Heberden nodes) B. Proximal IP (Bouchard nodes)
D. Soft tissue swelling: synovium & bursa		
1) Thickened synovium :		
↳ Site: over wrist & upper border of patella		
2) Bursa : traumatic cases or rheumatoid arthritis or gout		
↳ Sites : A. Prepatellar bursitis, "housemaid's knee" B. Infrapatellar bursitis, "clergyman's knee" C. Trochanteric bursitis, giving pain over lateral aspect of hip D. Olecranon bursitis, "student's elbow", characterized by pain and swelling in the elbow E. Subacromial bursitis, giving shoulder pain, is the most common form of bursitis.		

D. Surrounding structures:

- Muscle spasm in paraspinal muscles in ankylosing spondylitis
- Muscle wasting : in UL & LL : refer to neurology "wasting landmarks"

4. **Complication:** ulceration , rupture tendon (finger drop), trigger finger, carpal tunnel syndrome

5. **Gait & posture:** in LL & spine.

❖ N.B.: skin, joint contour, swellings, movement: can be examined as combined inspection & palpation.

2) Palpation:

1) Skin:

- For temperature : warmth (first at joint line then compare above & below):

- If temperature $\uparrow$ $\rightarrow$ activity

2) Joint contour for displacement (Subluxation), MCP, PIP, DIP, wrist

- If there is ulnar deviation $\rightarrow$ surely there is MCP subluxation.

3) Tenderness : at joint line (articular surface) عنك على وش العيان

A. UL	B. LL	C. Back
1. IPJ: by lateral compression, side to side 2. MCP J: metacarpal squeeze test for all or separately by 2 thumb from up & down 3. Wrist (Flexed): 2 thumb form above and below compression. 4. Elbow : from up & down 5. Shoulder : palpate around joint line خط القميص	1. MTP J: metatarsal squeeze test 2. Ankle joints: 2 thumb form above and below compression. 3. Knee joints: is elicited with the knee flexed 90° and the patient's foot resting on the table. The examiner's index & middle finger probes the meniscus along the joint line.	○ Palpate & percuss over the spine for tenderness: either by 2 thumbs in each vertebra or by your fist

4) Swellings:

A. SC nodules: special sites: see before.

- Character: Oval, variable size, firm, painless, mobile.

B. Effusion; fluctuant : in knee joint (In knee effusion search for Becker's cyst)

- Character: Cystic, has no edge with positive test for effusion:

Transpatellar fluctuation test (huge):

تخفي ركبته العيان مفرودة على الآخر
 اعصر من فوق الركبة متجها لأسفل علشان ترق السائل كله لجوه المفصل { لانه ممكن يكون راح لل supra-patellar pouch }
 ثم رقى بال index واستقبل بال thumb
 لو في massive effusion حتلاقي transmitted impulse من ال index لل thumb والعكس

Patellar tap (moderate):

تخفي ركبته العيان مفرودة على الآخر
 اعصر من فوق الركبة متجها لأسفل علشان ترق السائل كله لجوه المفصل
 دوس بال index & middle finger عموديا من فوق
 لو حسيت بعض ← no effusion
 لو حسيت بحاجة بتنزّل وتطلع ← moderate effusion

Milking or massage or bulge method (mild):

تخفي ركبته العيان مفرودة على الآخر
 رقى من فوق الركبة متجها لأسفل علشان ترق السائل كله لجوه المفصل وبعدين شيل ايدك (هناك بعض الدكتوراة يترك يده)
 فاضي السائل بايديك من ال medial compartment of the knee وبعدين ارجع رقى بايدك من ال lateral compartment
 لو في mild effusion حيطر bulge على ال medial compartment

C. Bony thickening:

- Character : Hard in consistency, fixed, on dorsolateral surface (never on palmer surface)
- In osteoarthritis : distal IP (Heberden nodes) & proximal IP (Bouchard nodes)

D. Thick synovial membrane by rolling technique by your fingers :

Character :

- Doughy sensation
- Firm in consistency (elastic or buggy or rubbery)
- Has an edge & non fluctuant swelling
- Site : wrist, upper border of patella
- DD from effusion :
- Effusion is cystic in consistency, no edge & has a special tests for effusion



❖ Inspection

○ Kyphosis



○ Lordosis



○ Scoliosis



○ Prepatellar bursitis



○ Olecranon bursitis



❖ Examination : Tenderness

↳ UL

○ IPJ



○ MCP J



○ Wrist



○ Elbow



○ Shoulder



↳ LL

○ MTP J

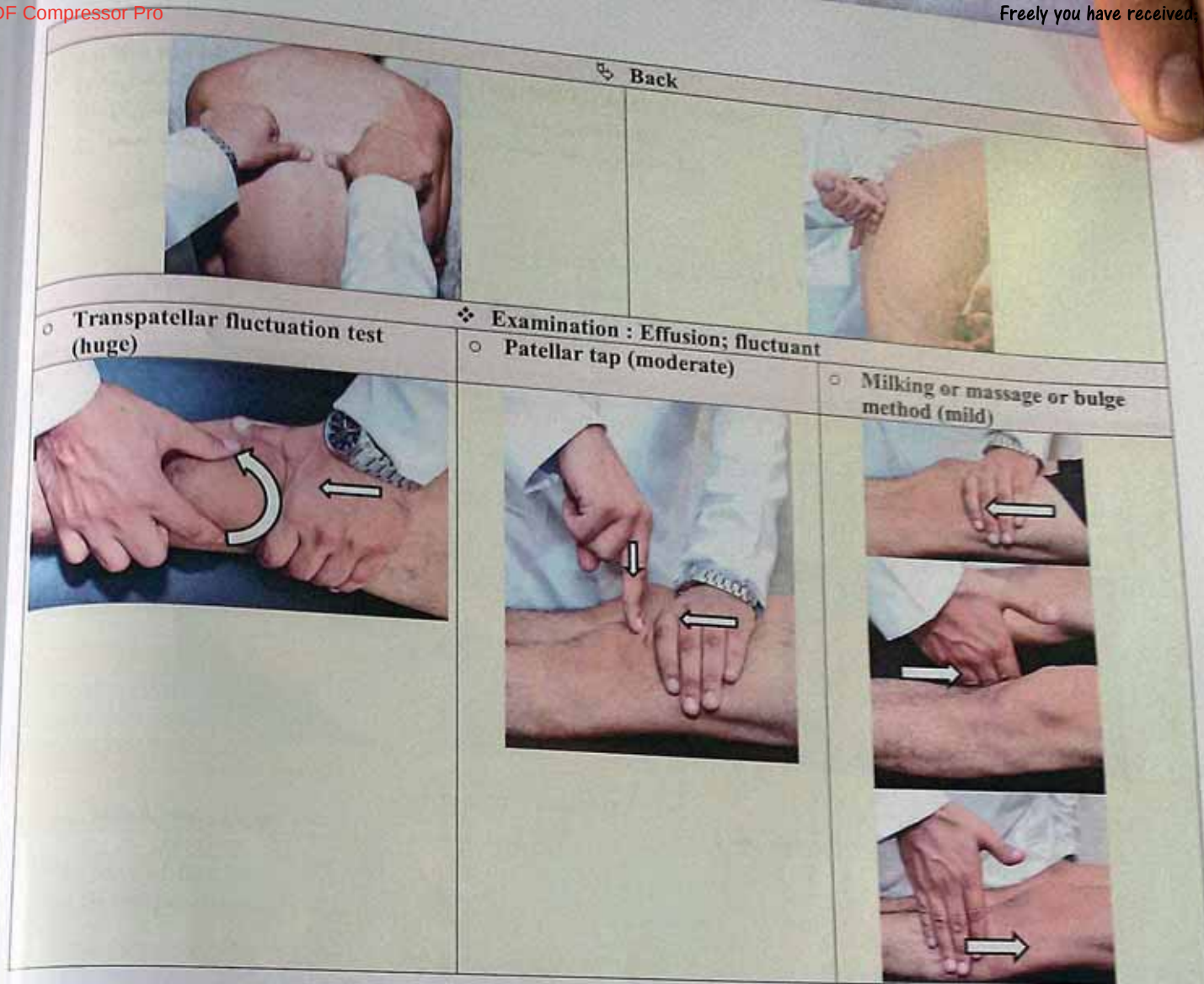


○ Ankle joint



○ Knee joint





3) Movement:

- To detect pain & tenderness
 - For crepitus: audible or palpable sound in the joint
 - Fine crepitus : not transmitted outside the joint (by stethoscope) → soft tissue pathology (capsule & synovium)
 - Coarse crepitation : transmitted outside the joint → bone & cartilage pathology (osteoarthritis)
 - Range of movement: active & then passive , accurately by goniometer
 - N.B.: Crepitus in RA is coarse tender crepitus due to 2ry osteoarthritis
 - In no movement (100%) limitation : ankylosing spondylitis
 - Limited in all direction in Rheumatoid arthritis (limitation of movement in active & passive to the same extent)
 - Abnormal in Charcot's joint "neuroarthropathy" : non-infectious destruction of bone & joints associated with neuropathy
- ❖ **Why do you examine movement passively after active examination?**
- Limitation in passive = limitation in active movement → arthritis
 - Passive move movement is better than active movement in:
 - Nerve affection
 - Myopathy
 - Rupture tendon



Goniometer

❖ Range of motion:

1. اطلب من العيان انه يعمل الحركة لوحده وشوف هيعمل الحركة للأخر ولا لا ← active
 ○ في مفاصل هتطلب من العيان يحركها زي ما اتعلمنا في ال neurology
 ○ وفي مفاصل أخرى هتطلب من العيان يعملها بطريقة معينة هبتم شرحها بعد قليل
2. بعدها الدكتور يعمل للعيان الحركة ← passive

ملاحظة: لما يقولك في الاوسكي افحص hand ولاقيتها RA لا تندفع ناحية فحص ال deformity
 ولكن افحص كل حاجة زي ما تعلمنا في ال General

❖ Peripheral joints:

1. Fingers: see neurology

- Flexion, Extension, Abduction, Adduction, Opposition

❖ How to determine functional state (capacity) of joint?

📖 **Fist test** : degree of power of fist = Hand grip
 "power grip"



📖 **Pinch test**: degree of power of opposition "precision pinch"



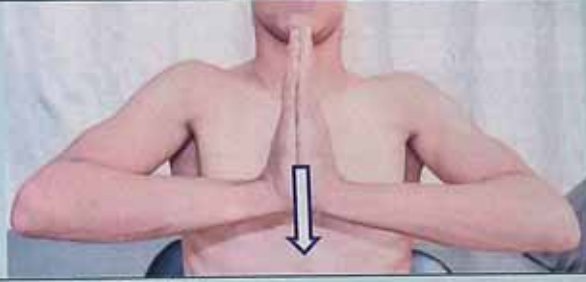
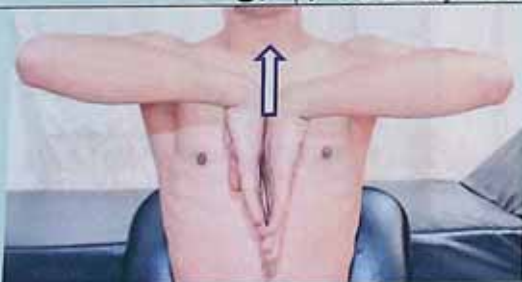
2. Wrist:

- Flexion = reversed prayer sign

○ ظهر ايديك في بعض وارفعهم لفوق

- Extension = prayer sign

○ بطن ايديك في بعض ونزلهم لتحت



- Radial deviation (20)

○ مد ذراعك وابعد ايديك

- Ulnar deviation (30)

○ مد ذراعك وضم ايديك



3. Radio-ulnar joint

- خلي العيان يحط ايديه جنبه وكوعه زاوية قائمة وال 2 thumbs باصين لفوق

- Supination (0-90°) كف ايديك للسقف

- Pronation (0-90°) كف ايديك للأرض



4. Elbow: see neurology

- Flexion

○ لازم ال forearm يلامس ال arm (150 درجة)

- Extension

○ لازم الكوع يتفرد 180 درجة

- Ask the patient to put both hands behind the head with elbows pointing laterally (flexion, abduction & external rotation)



5. Shoulder :

- Ask the patient to put the arms down and reach up behind the back (extension, adduction & full internal rotation)



○ Dorsiflexion (20°)



○ Planter flexion (30°)



6. Ankles & feet

○ Inversion (20°)

○ Eversion (10°)

- At Subtalar joint



7. Knee : see neurology

- Flexion: Ask the patient to flex each knee in turn and observe the range of movement (150°) and any signs of pain

- Extension: Ask the patient to straight each knee (180°), place a hand on the knee to feel the crepitus

8. Hip; Actively : ask the patient to do the action of hip; see neurology

- Passively: as following :

A. Flexion

- The patient flex his knee & move their hip into the flexed position as far as possible (120°)



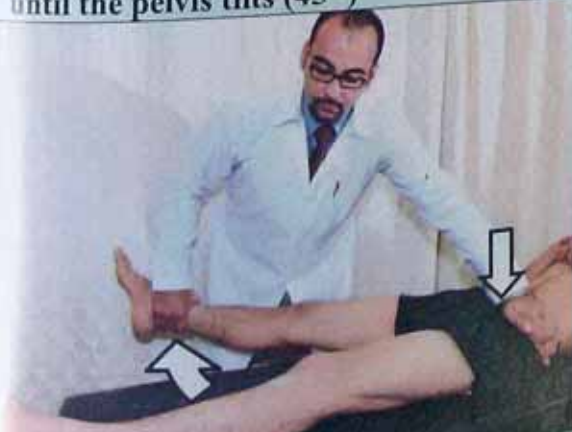
B. Extension

- The patient lie prone, immobilize the pelvis with one hand while extending the hip with the other hand (30°)



C. Abduction

- Make sure you stabilize the pelvis by placing a hand on the opposite ASIS and holding the ankle with the other hand. The hip is abducted until the pelvis tilts (45°)



D. Adduction

- Make sure you stabilize the pelvis by placing a hand on the opposite ASIS and holding the ankle with the other hand. Cross one leg over the other until pelvis begins to tilt (30°)



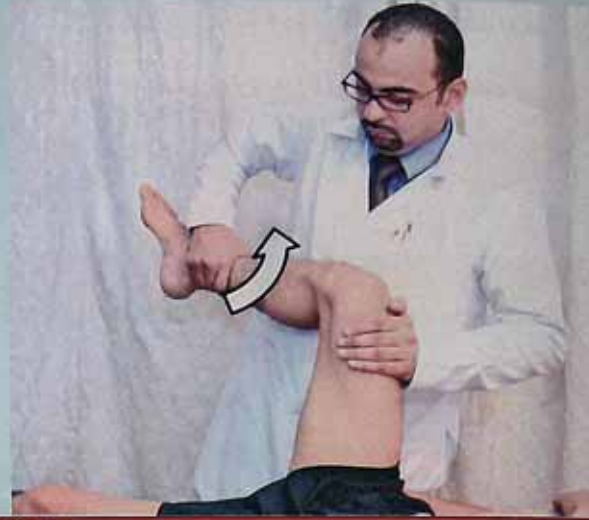
E. External rotation

- Flex the hip and knee to 90°, now move the leg medially (60°)



F. Internal rotation

- Flex the hip and knee to 90°, now move the leg laterally (45°)



❖ Axial joints

9. Cervical spine RA affect upper cervical

Flexion

- حاول تلمس صدرك برفقك



Extension: occiput to wall test

- وقف العيان, ظهره لارتق في الحيطه وقوله يحاول يلمس الحيطه براسه
- Normal = 0 (occiput to wall distance)



Lateral flexion (Rt& Lt): يحاول يلمس كتافه بودائه



Rotation (Rt& Lt): يلف رقبته شمال ويمين



10. Lumbar spine : OA affect cervical & lumbar

A. Flexion:

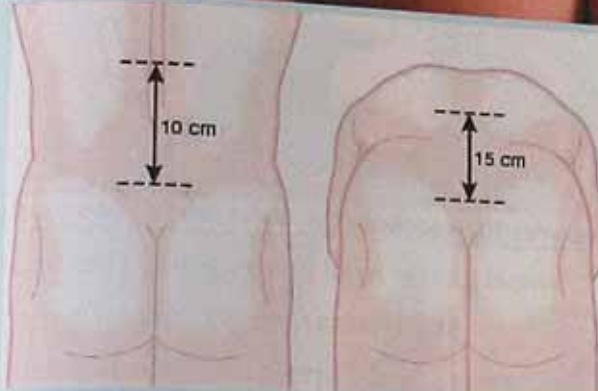
Finger to floor test



- ضم رجليك
- افرد ركبتيك
- حاول تلمس الأرض بصواعب إيديك
- قيس المسافة بين صواعبه والأرض : الطبيعي أقل من 5 سم

I. Schober's test: 2 points:

1. First at lumbo-sacral junction (Midway between PSIS; between 2 dimples of Venus)
2. Second above it by 10 cm



- خلي العيان يقف ويكشف ظهره
- حط نقطتين على ظهر العيان :
- الأولى : between 2 dimples of Venus
- الثانية : فوق الأول ب 10 سم
- قول للعيان يميل لقدام على قد ما يقدر
- قيس المسافة بين الخططين مرة أخرى

Normally 10 cm extends to 15 cm

If the distance < 15 cm → limitation

II. Modified Schober's test: 2 points:

1. First at lumbo-sacral junction (Midway between PSIS; between 2 dimples of Venus)
2. Above it by 15 cm

II. Modified Schober's test: 3 points

1. First at lumbo-sacral junction (Midway between PSIS; between 2 dimples of Venus)
2. Second above it by 10 cm
3. Third below it 5 cm



- خلي العيان يقف ويكشف ظهره
- حط نقطتين على ظهر العيان :
- الأولى : between 2 dimples of Venus
- الثانية : فوق الأول ب 10 سم
- الثالثة : تحت الأولى ب 5 سم
- قول للعيان يميل لقدام على قد ما يقدر
- قيس المسافة بين الخططين مرة أخرى

○ Normally: distance between the 2 line ≥ 15 cm

○ If the distance < 15 cm → limitation

B. Extension

C. Lateral flexion (Rt & Lt)

D. Thoraco lumbar Rotation (Rt & Lt)

- وقف العيان وخليه يرجع بظهره ورا على اليمين
- ما يقدر

- وقف العيان وقوله ايديك جنبك ... ويميل بجسمه يمين وشمال (كأنه بيحاول يزحلق ايده لرجله)

- قعد العيان علىشان تثبت ال Pelvis
- وقوله ببص لناعية كتفه بوسطه



11. Thoracic spine : see chest

- Ankylosing spondylitis affects thoracic spine
- Measurement by chest expansion by a tape at the level or below the nipple

❖ Special joints:

12. Temporomandibular:

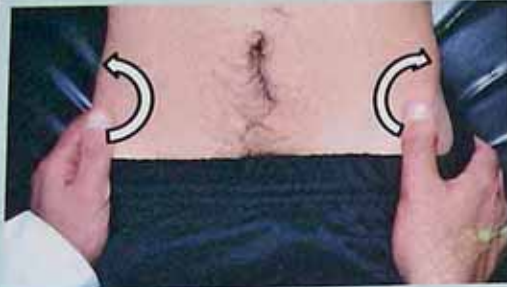
- Ask the patient to open and shut , protrusion and retraction , side to side

13. Sacroiliac joints

- Normally: pressure over Sacroiliac joint is painless
- If pressure → pain → sacroillitis

○ Pelvic springing :

- العيان نايم على ظهره
- حط إيدك على ال iliac bones 2 عند ال 2 ASIS
- حاول انك open the pelvis كأنه كتاب



○ Gaenslen's test:

- العيان نايم على ظهره
- رجل مددلة من السرير والرجل الشاقية مثبتة
- اضغط بإيدك على رجله اللي تحت واللي فوق
- خذ بالك انك كده بتفحص المفصل بتاع الرجل اللي مددلة من السرير



○ Lateral side compression of the pelvis



○ Direct pressure over the sacrum



Scleroderma

I. History:

- GIT : dysphagia, constipation
- Skin : limited finger mobility
- Joint: arthralgia
- Raynaud's phenomenon



II. Examination :

1. Skin:

- Early : edema : swollen & shiny skin due to edema
- Later: shiny, waxy, hard indurated , hairless, adherent to underlying structure, ± calcinosis, telangiectasia, ulcers & pigmentation (hyper or hypo)

❖ Types of scleroderma:



Morphea



A. Localized scleroderma= scleroderma without internal organ involvement :

- Morphea: circumscribed hard waxy plaque

B. Limited scleroderma = Acrosclerosis :

➤ Peripheral skin affection of :

- **Fingers of hands & feet** : swollen, shiny, limited mobility (sausage shape), pseudo-clubbing , digital infarcts, sclerodactyly
- **Face** : mask face, fish mouth, limited mouth opening , wide deformed nostrils with tipped nose & telangiectasia

Complications: CREST Syndrome: : Calccinosis, Raynaud's, Esophageal hypomotility, Sclerodactyly & Telangiectasia.

C. Progressive diffuse sclerosis :

- Diffuse skin lesion affecting trunk & proximal limbs as well as face, distal limbs (fingers)
- Severe internal organ damage and is generally more life threatening

D. Systemic sclerosis sine (without) scleroderma : rare

- Internal organ involvement without skin manifestation

E. Overlap syndrome (mixed connective tissue disease)

- Scleroderma associated with other autoimmune diseases

- This syndrome involves mixed features of at least 2 connective tissue disorders (SLE, Scleroderma , Polymyositis)

2. Joint examination: swollen, tender, limited mobility.

3. CVS : pericarditis, effusion, cardiomyopathy with HF, cor pulmonale (secondary to Pulmonary hypertension)

4. Chest: pleurisy, aspiration pneumonia , interstitial fibrosis.

5. CNS : headache & stroke during hypertensive renal crisis , myopathy

6. GIT : dysphagia , GORD

7. Renal: scleroderma renal crisis : malignant hypertension & CRF later on

❖ **Personal history:**❖ **Complaint:** pain in both hands, 10 years duration❖ **HPI:**

- The condition started 10 years ago by gradual onset and progressive course of **pain** which increase in early morning and decrease gradually with exercise, associated with **stiffness** that last for more than one hour & gradually resolves. The patient developed **marked hand and foot deformities** with loss of normal **joint function** in both upper and lower limbs.

- No symptoms suggesting **CVS** manifestations.

- No symptoms suggesting **chest** manifestations.

- No symptoms suggesting **Neurological or ocular** manifestations.

- No symptoms suggesting **anaemia**.

- No symptoms suggesting **renal** affection.

❖ **Past history:** No drugs, operation, disease (DM, HPN).❖ **Family history:**

- No consanguinity.

- No similar condition in family.

- No common disease in family.

❖ **General examination:**

- The patient is fully **conscious**, well oriented for time, place and person. Average mood and memory. The patient is co-operative with average intelligence.

- **Temperature:** 37.2° c.

- **Bl. Pressure:** 130/80.

- **Resp. Rate:** 16/minute, regular, average depth, abdomino-thoracic.

- **Pulse:** Regular, 72 beat/minute, average volume, no special character, equal on both sides, intact peripheral pulsation, vessel wall is not felt, no radiofemoral delay.

- **The patient** looks well, average built, no cyanosis, pallor or jaundice. He is lying free flat comfortable in bed.

- **Head & Neck :** joint exam (see later).

- **Upper limb:** No palmer erythema+ complete joint exam (see later).

- **Lower Limb:** No L.L. edema + complete joint exam (see later).

❖ **Local examination :****I. Peripheral joints**→ **By inspection:**1) **Deformities**

Big joints: in the form of flexion deformity in both elbows .

Small joints are affected in the form of:

- **Rt. Hand:** flexion deformity at MCP joint , Fusiform deformity in the middle finger, flexion deformity at PIP joint in the little finger.

- **Lt. hand:** Ulnar deviation of fingers at MCP joint , Swan neck deformity in index, subluxation at PIP joint in the middle finger, fusiform deformity at PIP joint in ring finger, flexion deformity at MCP joint in the little finger.

- **Both Feet:** show flexion deformity at PIP joints.

2) **Wasting**

- **UL:** There is bilateral wasting with guttering specially in the right hand . Both thenar eminences show flattening.

- **LL:** medial aspects of both thighs show loss of muscle bulk.

→ **By palpation :**

- **Synovium** isn't felt by rolling.
- Both elbows and middle finger of Rt. Hand show **subluxation**.
- Mild **tenderness** at wrist, MCP, PIP.
- **Coarse Crepitus** in both elbows and knees.

→ **By combined inspection & palpation :**

- **Skin:** no erythema, no palmar erythema, no psoriasis, no nail pitting, no scars, no warmth.
- **Swellings:** no evidence of bursopathy, no thickened synovium, no osteophytes, no S.C nodules, no PIP, MCP, wrist or knee effusions.

→ **Range of movement :**

- **UL:** Limitation of movement in both sides at all direction of movements both actively and passively:
 - **Fingers:** flexion, extension, abduction, adduction, opposition, hand grip.
 - **Wrist:** ulnar and radial deviation, flexion and extension
 - **Elbow:** extension and flexion.

- **LL:** normal range of movement.

II. **Cervical and lumbar vertebral column:**→ **by inspection :**

No deformity, no limitation of movement (flexion, extension, medial and lateral rotation, right and left tilting)

→ **By palpation:**

No tenderness over cervical or lumbar spines

III. **Special joints:**A. **Temporomandibular joint:**

- **Inspection:** no erythema, normal range of movement
- **Palpation:** no displacement, no crepitus

B. **Sacroiliac joint :**

- **No evidence of sacroiliitis**

Other Systems

- **CVS:** search for cardiomyopathy (Systemic congestion), pericardial effusion.
- **Neuro:** search for Entrapment neuropathy, atlanto-axial subluxation.
- **Hematological:** anemia or may be pancytopenia.
- **Chest:** search for pleural effusion, fibrosis, caplan's syndrome.
- **Renal:** search for nephrotic syndrome, CRF.
- **Ocular:** scleritis, Sjogren's syndrome.
- **Others:** myopathy, muscle wasting, HSM.

★ **Diagnosis**

- A case of chronic polyarthritis for DD most probably e.g. Rheumatoid arthritis

? **Why polyarthritis** → affecting of more than 4 joints group

? **Why chronic** → persisting for more than 6 weeks

? **Why rheumatoid :**

A. **Articular manifestation :**

- **Distribution**
- **Character:** morning stiffness more than 1 hour
- **Deformities**

B. **Extra-articular manifestation :**

- **SC nodules**
- **Mild multisystemic affection**

C. Type of patient :

- Sex : female
- Age: <40 years

D. Associated diseases :

- Sjogren syndrome (Sicca syndrome)
- Overlap syndrome

E. American collage of rheumatology criteria for diagnosis RA : see before

❖ How would you proceed to manage this case ?

1. Investigation of RA:

A. Investigation to prove diagnosis :

- Rheumatoid factor (non specific)
- Recent : anti CCP (specific) { Anti-citrullinated protein antibodies (ACPA) or anti-cyclic citrullinated peptide antibodies (anti-CCP)}
- X-ray for hands & feet

B. Investigation to detect disease activity :

- ESR
- CRP

C. Investigation to detect extra-articular (systemic) manifestations

2. Treatment :

- Anti-inflammatory: NSAIDs (aspirin, indomethacin, diclofenac,), Steroids.
- Disease-modifying drugs: Gold, D-penicillamine, Chloroquine, sulphasalazine, leflunomide.
- Immuno-suppressive drugs: Azathioprine, methotrexate
- Other measures: intra-articular injection of cortisone, surgical (arthroplasty, arthrodesis)

ENDOCRINOLOGY

ACROMEGALY

❖ **Etiology** : Excess growth hormone:

- 1) Child (before closure of epiphysis) : Acidophilic cells hyperplasia → gigantism
- 2) Adult (After closure of epiphysis): Acidophilic cells Adenoma → Acromegaly

❖ **History** :

1. **Height**:

- Tall stature in gigantism (disproportionate; span > height)
- Normal in acromegaly

2. **Enlargement** of skull, tongue (sleep apnea), prominent features, husky voice
3. Frequent **change** of hats, shoes, rings (tight)

4. Headache, blurring of vision, vomiting, ± convulsion, ± coma (pressure symptoms from tumor)

• الطاقم بصبوق عليه، الجزمة بصبوق عليه (بغيرها كل شهر)

• صداع بيزيد الصبح وترجيع مالوش علاقة بالأكل وزغلة

5. **DM symptoms**: polyuria, polydipsia, polyuria, ...

6. **Other systems** :

- Heart : HF manifestations رفرقة، ورم في رجلك، نهجان
- Chest : TB

- Abdomen : abdomen distention for organomegaly
- Neurological :

- PN & Myopathy بتحسن بتتميل أو شمشكة، بتقدر تشيل الحاجة أو تطلع سلالم
- Bitemporal hemianopia, 3rd, 4th 6th CN affection بيمشي بيخبط في الناس

- Bone & joints : osteoarthritis : pain increase with movement بتحسن بوجع في مفاصلك

❖ **Examination** :

I. **Vital signs** :

- BP: hypertension (salt & water retention + thick arteries)

II. **General overview** :

- A. Tall stature in gigantism (disproportionate; span > height)

- B. Head: Coarse features لو معاه صورة قديمة له قوله يطلعها وشوفها

1. Big elongated skull
2. Prominent bony process (frontal bossing & maxillary overgrowth)
3. Prominent supraorbital ridges, infraorbital puffiness
4. Big nose & ear
5. Thickened lips
6. Effect of DM on teeth, eye, skin, (see later)

7. Prognathism (prominent lower jaw) with widely separated teeth (DD from familial progeria)

→ N.B.: mandible is the only bone which is capable of growth till 40 years

8. Husky voice (thickened vocal cords)

9. True macroglossia : disproportion between mouth & tongue, complications :

- Traumatic ulcers (search for it)
- Difficult talking, Difficult deglutition, Dyspnea

❖ **Mechanism of macroglossia** :

- True macroglossia : Hypertrophy of tongue in Acromegaly
- Pseudo macroglossia : Deposition of tissues in myxedema

10. In nape area: wrinkled skin & folds, excess grease & sweats.

C. Upper, lower limbs:

- Thick hands (spade shaped)
- Enlarged with broad fingers, thickened blunted tips (sausage shaped)
- Big feet, prominent heel (calcaneal bone)

→ N.B.: Changes in head, UL, LL are marked in acromegaly & less prominent in gigantism



D. Skin changes :

- Skin tags in neck , may be associated with polyposis coli (precancerous)
- Skin is thickened, thrown into folds & wrinkled (lipolysis of subcutaneous fat)
- Greasy oily skin (seborrhea) ± sebaceous cyst
- Excessive sweating ± malodorous

E. Thyroid exam : goiter (GH ↑ thyroid hormone; euthyroidism)

III. Systemic examination :

1. CVS:

- Orthopnea due to HF as a result of HTN & Cardiomyopathy
→ *HF is the most common cause of morbidity & mortality in these cases.*
- Auscultate : weak Heart sounds & functional MR & TR

2. Chest:

- Auscultate for TB

3. Abdominal:

- Umbilical hernia , divarication of recti
- Visceromegaly e.g. hepatosplenomegaly (sonar finding)
- Colonic polyps & increased risk of colonic cancer
- Genital examination:

♂ In males :

- ♂ Gynecomastia & galactorrhea

♂ Sexually : 2 stage

- Early : increase desire & potency due to effect of GH

- Later: impotence due to :

- ✓ Prolactin like action

- ✓ Tumor destroys cells of Gonadotropin

- ✓ DM

♀ In females : menstrual troubles

4. CNS:

- A. Examine ocular CN (3rd, 4th, 6th CN) as result of pressure manifestation on optic chiasma especially field of vision (bitemporal hemianopia); confrontation test & acuity of vision.

- B. Examine for Proximal Myopathy

- C. Examine for sensation : peripheral neuritis

- Examine for Polyneuropathy (interstitial neuropathy) : feel for thickened nerves e.g. Ulnar nerve
- Examine for Entrapment neuropathy : carpal tunnel syndrome (median nerve compression) & tarsal tunnel syndrome (posterior tibial nerve compression) :



Skin Tags



Carpal tunnel syndrome



Tarsal tunnel syndrome

❖ Signs for diagnosis of carpal tunnel syndrome

- Inspection: wasting of the thenar eminence
- Motor power: abductor pollicis test: weakness of thumb movement (see neurology chapter)
- Sensory :
 - Sensory loss of the first 3 fingers & half of the ring finger
 - Numbness & pain reproduced by stressing on median nerve by either:
 - ♂ Tinel's sign: percuss with a hammer on flexor retinaculum
 - ♂ Phalen's sign : severe flexion of wrist for 1 minute عكس الهندي
- Feature of the cause e.g. Acromegaly, { others : Myxedema, DM, RA }

5. Joint : Osteoarthritis; knee crepitus, kyphosis



❖ **Diagnosis of acromegaly :**

- A case of acromegaly most probably due to pituitary adenoma (successfully surgically treated by trans-sphenoidal hypophysectomy)

❖ **Why acromegaly :**

1. Features : head, limbs ... ,
2. Signs of increased GH on skin, CVS, CNS, Abdomen, Gonads, ... etc.

❖ **Why pituitary adenoma :** local tumor manifestation (headache, field defect)❖ **Diagnosis of Gigantism :**

- A case of tall stature for DD most probably pituitary gigantism (successfully treated by irradiation)

❖ **Why pituitary :** evidence of increased growth hormone❖ **What are the DD of tall stature :**

- Constitutional : familial & racial
- Pituitary gigantism
- Hypogonadism
- Marfan's syndrome
- Klinefelter's syndrome
- Soto's syndrome (cerebral gigantism)

Cushing syndrome

❖ **Etiology:** ↑ cortisol due to :1. **Endogenous causes:**

A. Cortical tumor (20%)	B. Pituitary tumor (70%)	C. Para-malignant syndrome (10%)
• Cushing's syndrome	• Cushing's disease	
○ Abdominal mass	○ Micro-tumor → NO manifestation of ↑ ICT	○ Bronchogenic carcinoma ○ Ovarian neoplasm
○ ↓ ACTH (no pigmentation)	○ ↑ ACTH + Pigmentation	

2. **Exogenous causes; Cushingoid disease:** iatrogenic in patients taking cortisone in large amount for long duration e.g.:

- Rheumatic fever
- Bronchial asthma
- Nephrotic syndrome
- RA, SLE

❖ **History:**1. **Type of patient :** female, 30-40 years old, gradual onset, slowly progressive course:

- Excess weight gain (Obesity) تج زيادة وزن
- DM : polyuria, polydipsia, polyphagia تج عطش وجوع وبول كثير
- Hypertension: تج صداع وزغلة ووش في ودانك او نزيف من مناخيرك
- Skin changes
- Recent Pigmentation (Pituitary or Para-malignant causes)
- Depression → suicidal attacks
- Secondary myopathy : متقدرش تطلع السلم
- Osteoporosis: bone pain, pathological fracture
- Chest TB : ↓ resistance
- Decrease libido in both sexes
- Impotence in males
- Amenorrhea in females

2. **Past history:** History of drug intake & history of this disease : diseases treated by cortisone:

- Rheumatic fever, bronchial asthma, nephrotic syndrome, RA, SLE

3. **Family history :** obesity, hirsutism❖ **Examination :**1. **Vital signs :**

1. Hypertension "salt & water retention"
2. Fever 2ry to infection



II. General overview :

A. Stunted growth in children

B. Cushingoid feature :

1. Moon face i.e. rounded face with bloated cheeks (deposition of fat on the temporal & buccal regions)
2. Plethoric face : 2ry polycythemia
3. Trunkal obesity or centripetal obesity (samboxa shaped obesity):
 - Fat deposition in breast & abdomen
 - Not in buttocks → sunken buttocks
 - Thin limbs
4. Supraclavicular pad of fat
5. Buffalo hump (fat deposition in interscapular & dorsocervical region) + kyphosis



C. Effect of DM on teeth, eye, skin, (see later)

D. Skin changes :

- Thin skin : examine the skin fold on the dorsum of the hand (visible veins & cigarette paper sign)
- Skin ulcers, infections
- Vascular purpura & skin bruises
- Pigmentation (Pituitary or Para-malignant causes)
- Stria rubra (search for it in arms , breast, abdomen , thighs)
- Stria alba because of rapid ↑ increase of abdomen
- Effect of DM on skin, (see later)
- Hirsutism in females, Baldness in males,
- Acne Vulgaris in both sexes.



III. Systemic examination :

1. CVS:

- Hypertensive complications (LV enlargement + LVF)
- Rheumatic fever (as a cause)

2. Chest:

- Bronchogenic carcinoma, bronchial asthma (as a cause)
- Full examination for infections especially TB (crepitation & bronchial breathing)

3. Abdominal:

- Skin changes (see before), Bleeding tendency (vascular purpura)
- Pot belly abdomen, gynecomastia, Fatty liver
- Adrenal cortical tumors may be palpated as abdominal mass; suprarenal mass (as a cause)
- Genital examination :



Male	Female
• Decrease libido in both sexes	
• Impotence	• Amenorrhea

4. CNS:

- Examine mentality for psychosis mainly depression
- Muscle wasting & Secondary myopathy (Weakness of LMNL)
- DM: neuropathy : peripheral neuritis : superficial sensory loss, deep sensory loss (ataxia)

5. Joint :

- Osteoporosis : tender spine , deformities
- Stunted growth in young children.

❖ **Diagnosis:** a case of obesity most probably Cushing's syndrome, most probably due to ... (e.g. pituitary tumor in case of pigmentation)

Or

❖ A case of obesity with DM & HTN most probably cushingoid due to treatment

❖ **DD between** Cushing & simple obesity associated with HTN & DM

- Fat distribution & Specific associations : striae rubra, purpura , myopathy, osteoporosis

Addison's disease (Hypocorticism)

Etiology :

1. Surgical: Removal of supra-renal cortex
2. Irradiation
3. Diseases : auto-immune & TB, sarcoidosis , hemochromatosis

History:

- | | |
|---|---|
| <ul style="list-style-type: none"> • Asthenia, Marked weakness, fatigue, decreased weight
تعب هائل و بطن ، همدان ، ما يعرفش بتحرك من مكانه • Hypotension : Craving for salts
"pathognomonic"
ليس له أعراض ماعدا يتوحم على الملح • Generalized pigmentation; tanning of skin
لونه يغمق • GIT upset: anorexia, nausea, vomiting, diarrhea, polyuria | <ul style="list-style-type: none"> • Postural hypotension up to syncopal attack
لو قعدت أو قمت فجأة ادوخ • Marked hypoglycemia : craving for eating especially sugar
Headache, blurring of vision, drowsiness , hunger pain
صداع وزغللة ودوخة وبطنه توجعه • Evidence of TB : <ul style="list-style-type: none"> - Chest: history of TB in secondary Addison - Pain in the renal angle in TB nephritis |
|---|---|

- History of adrenal crisis : shock , hospitalization & fluid replacement.
- Past history : History for operation adrenalectomy for treatment of Cushing's syndrome

Examination :

Vital signs :

- Marked hypotension :
 - Never diagnose Addison's with systolic blood pressure more than 110
- Postural hypotension:

قيس الضغط مرتين : مرة وهو نايم ومرة وهو واقف : هتلاقي :

- Systolic blood pressure decreased by ≥ 20 mmHg or Diastolic blood pressure decreased by ≥ 10 mmHg

General overview :

- A. Weight loss
- B. Skin changes :
 - Vitiligo in autoimmune
 - Generalized Hyperpigmentation :

Sites & color :

Skin : Tanning (dark brown)



II. Mucous membrane : Slate coloured بنفسجي محمر



1. Exposed to sun light :
 - Face, neck, neck lace, Dorsum of hands
2. Pressure areas:
 - Elbow , knee, extensor surface of limbs
3. Friction area :
 - Axilla, inner thigh
4. Previously pigmented area became more pigmented :
 - Areola, nipple , umbilicus
5. Scars (recently only)
6. Hands:
 - Palmar : palmer creases
 - Dorsum : knuckles , nail folds

1. Inner side of lips
2. Gums
3. Buccal mucosa i.e. inner side of cheeks

⚠ DD with Peutz-Jeghers syndrome: benign **hamartomatous polyps** in the gastrointestinal tract (GIT bleeding) and **hyperpigmented macules** on the lips and oral mucosa

4. Palate
5. Tongue
6. Rectal mucosa (by proctoscope)

III. Systemic examination :

1. Chest: to exclude TB (fibrosis, cavity or infection)
2. Abdominal:
 - Examine for loss of pubic & axillary hair in secondary forms of adrenal insufficiency
 - Renal angle tenderness in miliary TB
 - Scar of operation of adrenalectomy

❖ Diagnosis :

- A case of hyperpigmentation with asthenia & hypotension for DD most probably Addison's most probably due to TB

❖ Why Addison's :

1. Distribution of pigmentation specially MM
2. Specific association : postural hypotension

- ❖ **DD of Addison's** = causes of generalized pigmentation : see general chapter

Diabetes mellitus

❖ History :

- Polyuria & nocturia
- Polydipsia (excessive thirst)
- Polyphagia + weight loss
- Pruritis (monilia)
- Pains & paresthesia : Peripheral neuritis
- Premature loosening of teeth
- Blurred vision
- Impotence , urinary incontinence
- Ask about time (onset) of development of DM : type I, type II
- Ask about symptoms of other systems "*complications*"
- Ask about family history
- Ask about secondary causes :
 - Pancreatic causes : chronic pancreatitis
 - Endocrinal causes : ↑ anti-insulin e.g. Cushing, pheochromocytoma
 - Others : liver cirrhosis - drugs (steroids - thiazide) - Friedreich's ataxia



Thirst



Excess urine



Hunger



Blurred vision

❖ Examination :

I. Vital signs :

1. Temperature for infection
2. Respirator rate : Kussmaul breathing: deep rapid breathing (Air hunger), Acetone smell in DKA
3. Blood pressure:
 - BP hypertension (early atherosclerosis)
 - Postural hypotension due to autonomic neuropathy : Measure BP both erect & supine
4. Pulse :
 - Rapid weak in dehydration
 - Rapid strong in hypoglycemia
 - Peripheral pulse for ischemia

II. General overview :

A. Body :

- Appearance : emaciated in long standing DM
- Built: Under built in IDDM , overbuilt in NIDDM
- Conscious level : coma, drowsiness in hypo or hyperglycemia
- Decubitus: orthopenic in left sided HF secondary to diabetic cardiomyopathy
- Expression : as an association e.g. Cushing syndrome

B. Head & neck :

- Eye : Xanthomatosis , blepharitis, conjunctivitis , cataract
- Mouth : beefy tongue , gingivitis, dental caries, loosening of teeth & dry tongue & acetone odor in mouth
- Neck vein : for congested pulsating neck vein in cardiomyopathy heart failure



Charcot joints

- C. Upper, lower limbs:
- Dupuytren's contracture
 - Trigger finger: when you try to straighten your finger, it will lock or catch before popping out straight
 - Charcot joints "neuroarthropathy": non-infectious destruction of bone & joints associated with neuropathy
 - Wasting of small muscles of the hand
 - Tremors in hypoglycemia, sweaty hands, interdigital fungal infection in nails, trophic changes or ulcers (2ry to neuropathy)
 - LL oedema, trophic changes, ischemia (pain, pallor, coldness, intermittent claudication)
 - Monofilament test:
 - A standardized filament is pressed against part of the foot.
 - When the filament bends, its tip is exerting a pressure of 10 grams (therefore this monofilament is often referred to as the 10 gram monofilament).
 - If the patient cannot feel the monofilament at certain specified sites on the foot, he/she has lost enough sensation to be at risk of developing a neuropathic ulcer.
- D. Skin changes:



○ Diabetic dermopathy	○ Necrobiosis lipoidica diabeticorum	○ Bullosis diabeticorum	○ Acanthosis nigricans
			
○ Red painless papules over shin of tibia & may ulcerate, due to occlusion of the cutaneous blood vessels	○ Red painless papules with yellow center over shin of tibia & may ulcerate, due to microangiopathy & fat deposition	○ Non inflammatory, spontaneous, painless blister, often in acral locations (peripheral body parts, such as feet, toes, hands, fingers, ears or nose)	○ Hyper pigmented (brown to black) velvety patches over the neck, axilla & ano-genital region, due to insulin spillover into the skin (due to excess insulin in insulin resistance)

- Vitiligo
- Infections e.g.
 - Carbuncles & furuncles in the back
 - Monilia infection (pruritus): under breast, axilla, buttocks, groin
- Delayed healing of wounds



- Xanthomata
- Carotinema
- Cutaneous features of diabetic foot e.g. colour changes, trophic ulcers & gangrene
- Complication of treatment e.g. Insulin injection sites & insulin lipodystrophy (atrophy of SC fat at sites of injections forming skin dimples)

II. Systemic examination:

1. CVS:
- Anginal pain, Accentuated S1 (hypertension), cardiomyopathy: dilated enlarged heart
 - Chest: effusion, cavity, fibrosis, for infection esp. TB, (TB follows DM as its shadow)
2. Abdominal:
- Fatty liver, Diabetic glomerulosclerosis
 - Dyspepsia, nausea, vomiting, diarrhea, abdominal pain (DKA)
 - Liver manifestation: pain & gall stones (chronic cholecystitis)
3. Genital examination:
- Impotence in males
 - Sterility, abortion, premature labour, big babies in females
4. NS: see neurology chapter

Tetany

❖ **Normal calcium level:** 8.5 – 10.5 mg%

1. Ionized (active): 50%
2. Non ionized (reserve): 50 % :
 - 40 % bound to albumin
 - 10 % any other bond (Ca carbonate, phosphate,)

❖ **Tetany** : state of ↑ neuro-muscular irritability due to ↓ ionized Ca, ↓ Mg or alkalosis

❖ **History:**

1) **Hypocalcemia** :

- Hypoparathyroidism : Thyroid surgery (neck scar), Neck irradiation
- Mal-absorption syndrome, Acute pancreatitis , Chronic renal failure

2) **Hypomagnesaemia** : excess diuretics , mal-absorption syndrome

3) **Alkalosis** :

- | | | |
|------------------------------------|------------------------------------|---------------------|
| ○ Hypokalemia e.g. Conn's syndrome | ○ Excess antacid in duodenal ulcer | ○ Repeated vomiting |
|------------------------------------|------------------------------------|---------------------|

❖ **Examination :**

I. **Vital signs** :

- BP measurement precipitate to carpal spasm "Trousseau sign", So Ca⁺⁺ is given before
- Hypotension 2ry to hypocalcemia

II. **Clinical picture of latent tetany** : serum Ca = 7-9 mg%

❖ Manifestations of tetany are absent

❖ Manifestations appear by provocative tests:

A. Chvostek's sign :

- Tapping over facial nerve (in front of ear) causes contraction of facial muscles
- Tapping over peroneal muscle causes contraction of peroneal ms (peroneal sign)

B. Trousseau's sign : ↑ BP above systolic on brachial artery for 3 minute causes carpal spasm

C. Erb's sign : Current < 4 milli-amperes causes muscles contraction (normally 8 m. Amp)

III. **Clinical picture of manifest tetany** : serum Ca < 7mg %

A. Nerves :

- Irritability & Parasthesia : numbness & tingling in perioral area, fingers & toes

B. Muscles : muscles twitches & convulsion

❖ Muscles spasm :

○ CARPOPEDAL SPASM:

- Carpo : flexion of wrist, flexion of MCP joint & extension of PIP & DIP joints, thumb adducted
- Pedal : planter flexion & inversion

○ Eye: dryness, blepharospasm

○ Facial & master muscles : risus sardonicus & trismus (lock jaw)

○ Laryngeal muscles : laryngismus stridulus

○ Diaphragm : hiccough

○ GIT : abdominal colics

○ Back muscles : opithotonous position

C. All provocative tests are +ve & hyperreflexia

D. Ectodermal changes in hypoparathyroidism only :

- Teeth: hypoplastic, transverse furrows & punctate holes if the condition started before formation of permanent teeth
- Skin: dry, rough, atrophy, ulceration & monilia infection
- Loss of hair & alopecia
- Nails: brittle + striated, loss of luster
- Lens : cataract

❖ **Diagnosis** :

- Recurrent cramps of hands & feet for DD most probably tetany due to hypoparathyroidism



Dwarfism, Stunted growth, Short Stature

- ❖ **Height** : is measured from growth curve (it ranges between 3rd & 97th percentile of same age & sex)
- A. The height and span are equal (proportionate) :
 - The height : distance from occiput to heel in upright position:
 - The head is positioned properly : neutral position & looking straight
 - The shoulders, buttocks & heels touching the measurement surface
 - Arms at sides
 - Legs straight & knee together
 - Feet flat, The heels are almost together
- Span : the distance between the tip of middle fingers with outstretched hands
- B. Lower and upper segment are usually equal :
 - Upper segment : distance from the occiput to the symphysis pubis
 - Lower segment : distance from symphysis pubis to the floor



❖ Short stature "dwarfism":

- Height below 3rd percentile of normal people of their own age & sex
- Short stature + lack of sexual development = infantilism

❖ Causes of dwarfism :

1. Chronic illness :

- Bilharziasis
- Chronic heart disease : F4
- Chest disease : polycystic lung
- Chronic nephritis

2. Endocrine:

- a) Familial & constitutional : the commonest
- b) ↓ Growth hormone :
 - Pituitary dwarfism : Levi-Lorain syndrome: Proportionate dwarfism + hypogonadism
 - Hypothalamic syndrome: Frohlich's syndrome : hypogonadism + obesity + somnolence, diabetes insipidus, hyperphagia
- c) ↓ T4 = cretinism : mental retardation, stunted growth & disproportionate dwarfism (span > height)
- d) ↑ Sex hormones : precocious puberty (premature fusion of epiphysis).
- e) ↑ Cortisol : Cushing syndrome or excess steroid treatment (cortisol block the ability of GH to produce somatomedin, a hepatic peptide responsible for bone growth)
- f) ↑ Blood sugar : DM since childhood

3. Skeletal disorder:

a) Acquired:

- Rickets
- Pott's disease TB spine may end in severe kyphoscoliosis & dwarfism

b) Genetic:

1. Achondroplasia :

- Limbs: short
- Trunk : normal
- Face is small compared with vault of skull , sexual development normal

2. Osteochondrodystrophies: limbs & trunk : short & deformed

3. Osteogenesis imperfecta :

- Bones are flattened, deformed due to repeated fractures
- Blue sclera & conductive deafness

4. Turner's syndrome : dwarfism + primary amenorrhoea, webbing of neck.

5. Mongolism :

- Mental deficiency , Narrow eye - slits which are also sloped outwards & Deep fissured tongue



Myxedema; hypothyroidism in adults

❖ Etiology :

A. Consequence of thyrotoxicosis :

- Drug treatment by anti-thyroid drugs
- Radioactive iodine
- Surgical : subtotal thyroidectomy

B. Non - Consequence of thyrotoxicosis:

- Primary atrophy: idiopathic (commonest)
- Secondary myxedema : due to pituitary failure
- Auto-immune : Hashimoto thyroiditis (& vitiligo)

❖ History :

❖ **Type of patient** : female, 30-40 years old, gradual onset, slowly progressive course

1. History of the cause : drug intake, radioactive iodine & surgical treatment
2. Intolerance to cold بتحبي الشتاء ولا الصيف : هتقولك بحب الصيف علشان بيرد أوي في الشتاء
3. Weight gain, loss of appetite
4. Hypertension
5. Hypoglycemia
6. Asthenia : Tiredness, weakness
7. Slow thinking, apathy, poor memory, hypersomnia
8. Constipation لازم حقنة شرجية علشان أعمل حmam dyspepsia
9. Genital:
 - Female : menorrhagia, galactorrhea
 - Male : impotence, gynecomastia

❖ Examination :

I. Vital signs :

- Temperature: hypothermia (normal if treated by thyroxin)
- Pulse : bradycardia (normal if treated by thyroxin)
- BP : hypertension (normal if treated by thyroxin)

II. General overview :

A. Sign for cold intolerance : heavy clothes

B. Overbuilt : weight gain

C. Head:

- Appearance : Puffy face, expressionless, bloated , malar flush
- Hair : thin, dry, alopecia
- Puffiness of upper eye lid due to periorbital edema
- Eyes : sparse hair, loss of outer 1/3 of eye brow + cataract
- Xanthlasma
- Mouth :
 - Thick lips
 - Pallor due to :
 - Anemia (all types),
 - V.C.
 - Myxedematous deposition
 - Carotinema → ↓thyroxin which metabolize carotin
 - Husky voice due to deposition of myxedematous tissue on vocal cords
 - Pseudo macroglossia (thick, protruded) : disproportion between mouth & tongue, complications :
 - Traumatic ulcers (search for it)
 - Difficult talking, Difficult deglutition, Dyspnea

D. Upper, lower limbs:

- Non pitting oedema due to intradermal accumulation of proteins



E. Skin changes :

- Cold, yellowish discoloration due carotenemia , pale due to anemia
- Thickened skin, non-sweaty
- Dry, rough, scaly
- Vitiligo in autoimmune cases
- Thick nail, hair is sparse

F. Thyroid exam : For etiology

- Scar in previous operation: thyroidectomy
- Firm, diffusely enlarged : Hashimoto's
- ↓ in idiopathic atrophy

III. Systemic examination :

1. CVS:

→ CVS is the commonest cause of mortality

- Hypertension
- Sinus bradycardia
- Pericardial effusion

○ Coronary heart disease but without symptoms of angina:

- ↳ Why asymptomatic ? → Inactive heart & inactive patient
- ↳ When symptoms appears ? → Symptoms appear with treatment, so increase dose very gradually

○ Cardiomegaly : cardiomyopathy - cholesterol pericardial effusion

2. Chest:

- Auscultate for pleural effusion

3. Abdominal:

- Constipation → abdominal distention → stria alba
- Enlarged liver "fatty liver"
- Genital examination:

- Normal genitalia (no atrophy) & Normal 2ry sexual hair (suprapubic & axillary)

4. CNS:

A. Mentality :

- Slow cerebration, apathy
- Slow movement , slow response to stimuli, slow talking, slow thinking , decrease memory
- Myxedema madness : psychosis , depression

B. Speech : slurred speech, hoarse voice

C. Conductive deafness due to deposition of myxedematous tissue in auditory canal

D. Examine for Proximal Myopathy

E. Examine for sensation : peripheral neuritis

- Examine for Polyneuropathy (interstitial neuropathy) : feel for thickened nerves e.g. Ulnar nerve
- Examine for Entrapment neuropathy : carpal tunnel syndrome & tarsal tunnel syndrome:

❖ Signs for diagnosis of carpal tunnel syndrome : see before.

F. Hyporeflexia : Delayed relaxation time of reflexes i.e. suspended jerk

5. Joint : Osteoarthritis; knee crepitus

❖ Diagnosis :

A case of obesity with HTN & neurological manifestation most probably myxedema , most probably autoimmune

❖ Why : myxedema:

📖 From history & examination:

- A. Evidence of thyroxin deficiency
- B. Evidence of deposition of myxedematous tissue
- C. Specific : delayed relaxation time of reflexes

❖ Why autoimmune :

- A. Exclude iatrogenic causes
- B. Associated endocrinopathies e.g. Dm
- C. Trial for corticosteroid



Sheehan's Syndrome

❖ Definition :

- Simmond's disease : Panhypopituitarism due to any cause
- Sheehan's syndrome : Panhypopituitarism due to post partum hemorrhage "post-partum pituitary infarction"

❖ Pathogenesis :

- At the end of pregnancy, blood is hypercoagulable but blood vessels dilated → no thrombosis
- Post partum hemorrhage → ↓ blood pressure → V.C. → coagulation → pituitary infarction → loss of anterior pituitary function
- Posterior pituitary is not affected because blood flow increase to posterior pituitary for lactation

❖ History & Clinical picture:

- Type of patient : female in child bearing period
- I. Evidence of hypogonadism (↓ gonadotrophic hormones (FSH, LH); secondary hypogonadism):
 1. In females: History of labor with post partum hemorrhage followed by failure of lactation (due to ↓ prolactin) + amenorrhea, breast atrophy
 2. In males : impotence
 3. In both sex: loss of pubic & axillary hair & loss of libido, loss of fertility, atrophy of genitalia
- II. Evidence of hypocorticism (↓ ACTH; secondary adrenal failure):
 4. Loss of sun tanning power

لهم مهما اتعرضت للشمس يفضل لونها زي ما هو ما تسمرش

5. Depigmentation of body especially areola (↓ MSH)

لهم عكس المتوقع من واحدة لسه والد

6. Hypotension is not severe due to continued release of Aldosterone

III. Evidence of hypothyroidism (↓ TSH; secondary myxedema) :

7. Symptoms of myxedema {but minimal weigh gain} : cold intolerance, ...

❖ N.B.

A. GH deficiency : not clinically detectable in adults; just:

- Fine wrinkles & weakness
- ↑↑↑ sensitivity to insulin : hypoglycemia

B. Due to cortisone treatment : the patient is cushingoid → DM عكس المتوقع

C. Diabetes insipidus very late if posterior pituitary affected

- تقولك بشرب حوالي 15 لتر يومياً وبروح الحمام أنزل مياه كتير جداً وبإخذ علاج بخاخه Synthetic vasopressin puff

- ❖ **Diagnosis :** a cases of 2ry amenorrhea for DD most probably panhypopituitarism, (Sheehan's syndrome), She is under cortisone replacement therapy

❖ Why Pituitary (Sheehan's) ?

- Evidence of hypogonadism
- Evidence of hypocorticism
- Evidence of hypothyroidism

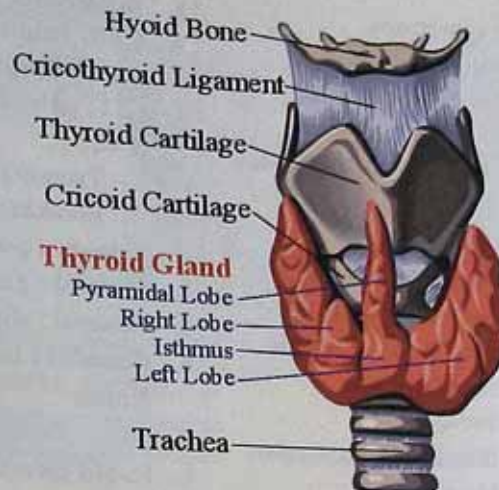
❖ DD: myxedema :

- N.B. : 3 differences between myxedema & Sheehan's:
- Etiology
- Manifestation: decrease gonadotropin (Gonadal affection), & decrease ACTH
- Treatment : cushingoid features

لهم أهمية التفريق : لأنها لو اتشخصت myxedema هيدوها thyroxin ← Addisonian crisis

GOITER

- **Normally** : thyroid gland is not felt , if enlarged → Goiter
 - ❖ **Classification** :
1. **Simple goiter** :
 - Simple physiological goiter
 - Simple colloidal goiter
 - Simple nodular goiter (SNG)
 2. **Toxic goiter**:
 - Primary or secondary toxic goiter
 - Toxic nodule
 3. **Neoplastic** :
 - Benign neoplasm
 - Malignant thyroid
 4. **Inflammation** as Hashimoto's thyroiditis
 5. **Congenital** as Cretinoid goiter



GOITER HISTORY

A. Personal History

1. Name : الإسم ثلاثي	2. Age : ○ Toxic goiter: - 1ry between 20 – 30 years - 2ry between 30 – 50 years ○ Malignancy above 50 years ○ SNG at any age	3. Sex : ○ Females : simple goiter ○ Males : malignancy
4. Occupation : ○ Exposure to radiation : malignancy	5. Marriage : for infertility or impotence : toxic goiter 6. Menstrual history if female for disturbance	7. Residence & address : for endemic area as Fayoum or Wadi El-Natroun
8. Habits of medical importance.		9. Handedness .

B. Complaint + duration: إي اللي جابك المستشفى ؟

Common complaints :

- Neck swelling
- Symptoms of thyrotoxicosis (see later)
- Symptoms of malignancy (see later)

C. History of present illness = HPI المرض بدأ معاك إزاي , آخر مرة كنت كويس فيها إمتى ؟

1. Analysis of the complaint : swelling ± pain	2. Pressure symptoms (local complications)
3. Systemic symptoms (general complications)	
4. Investigations & treatment received	

I. Analysis of the complaint : swelling ± pain A. Analysis : OCD, site, number, associated other swelling as LNs metastasis B. Pain if present : - OCD, ↑, ↓, Associated symptoms - Character, Site , Radiation - Relation to (meal, respiration, exertion, postural)	III. Systemic symptoms (general complications) A. Toxic manifestations Metabolic: - Loss of weight inspite of good appetite - Intolerance to heat, Excessive sweating CVS: Palpitation even at rest Chest: dyspnea GIT: polyphagia ± diarrhea CNS: - Tremors of tongue & hand , Irritability & insomnia - Weakness of proximal limb muscle Urinary : polyuria Skeletal : generalized bone ache General : diplopia of eye Gonadal : impotence in male, menstrual disturbance in female
II. Pressure symptoms (local complications): Trachea → Dyspnea Esophagus → Dysphagia but rare Sympathetic chain (Homer's syndrome) = (Ptosis, myosis, enophthalmos, anhydrosis) Internal jugular vein → Oedema of eye lid Carotid artery → Postural fainting Recurrent laryngeal nerve : → Hoarseness of voice if unilateral affection → Stridor if bilateral affection	B. Malignant manifestation : Rapid increase in size with short duration. Pain is related to swelling or referred to ear Metastasis to Lung, Brain, Liver, Bone Symptoms of infiltration (see pressure symptoms)
IV. Investigations: Thyroid function tests, Radioactive iodine study, U/S, Biopsy ❖ Treatment : Medical, surgical, radiotherapy	

D. Past history :

- Similar condition, Diseases as DM, hypertension, heart disease etc.
- History of drug allergy or goiterogenic drugs as Thiouracil, neomercazole
- History of neck irradiation to exclude risk of malignancy.
- History of neck operation as thyroidectomy or L.N biopsy

E. Family history

- Similar condition as in endemic goitre
- Cancer thyroid as medullary carcinoma, Congenital: Pendred's syndrome

Discussion on history

1. What are the causes of painful goitre ? Malignancy , acute thyroiditis & hemorrhage in cyst	2. Why is dysphagia as pressure symptom being rare ? Because oesophagus is a muscular tube.
3. What are other causes of Horner's syndrome ? - Compression by goitre, pan-cost tumor & carotid aneurysm. - Complication after cervical sympathectomy. - Injury of lower part of brachial plexus.	4. What are the causes of loss of weight inspite of good appetite ? - Toxic goiter, - Uncontrolled D.M., - Parasitic infestation (hydatid cyst) - Mal-absorption syndrome.
5. What are the causes of polyuria in case of toxic goitre ?	
Secondary DM → Glucosuria.	↑ Metabolic rate ↑ Intake of water 2ry to polyphagia ↑ COP → ↑Renal blood flow → ↑ GFR
6. Why is pain in malignancy of thyroid referred to Ear ? Because of ear has same dermatomal supply i.e. Arnold nerve	
7. What are the types of biopsy done in case of goitre ? FNAC, True cut biopsy & excisional biopsy.	
8. What are symptoms of Pendred's syndrome ? Goitre, dwarfism & deafness.	

I. General examination:

A. Vital signs:

- Temperature : ↑ With toxic goitre.
- Pulse rate : with Toxic goiter
- Tachycardia, regular or irregular , large volume, equal on both side & water hammer pulse as special characters
- Blood pressure : High systole & low diastole
- R.R may be increased

B. General examination :

❖ General overview:

• Appearance:

- Ill with cachexia as in malignancy
- Irritable & alert as in toxic goiter
- Built: under built as in hyperthyroidism or malignancy
- Conscious: conscious but apathy as in hypothyroidism
- Decubitus: orthopnea as in HF
- Facial Expression : starring look as in toxic goiter
- Intolerance to hot weather

❖ Systemic overview :

1. Head:

- Skull for swellings as (bone metastasis).
- Lip :
- Pallor : thyroid dysfunction (hyper or hypo) or malignancy
- Cyanosis : huge retro-sternal goiter
- Mouth for ectopic thyroid.
- Tongue for tremors. {N.B: Tongue must be unsupported}
- Eye for
 - Jaundice : anti-thyroid drugs or liver metastasis
 - Eye signs of thyrotoxicosis : see later
 - Exophthalmos: actual protrusion of eye ball, examination : see later

2. Neck : see local examination

3. Upper limb :

- Palmer erythema
- Plummer's nails: distal onycholysis
- Fine tremors of hand
- Warm, Moist, sweaty hands if Thyrotoxicosis. {N.B. If cold = psychoneurosis}



- Thyroid acropachy: in 1 % of patient with Graves : exophthalmos + pretibial myxedema + clubbing

Lower limbs :

- Edema pitting if HF
- Pre-tibial myxedema
- Dorsalis pedis pulsation



❖ Sites of tremors :

- Outstretched hand
- Slightly closed upper eye lid
- Protruded unsupported tongue



5. CVS:

- Sinus tachycardia (all types of arrhythmias except heart block)
- High COP failure, Flow murmurs

6. Chest: metastasis to chest wall & mediastinal syndrome

7. Abdomen : liver & spleen enlargement

8. CNS:

- Myopathy, Hyper-reflexia, Fine tremors ,nervousness & irritability

9. Back : for metastasis

How to examine exophthalmos :

I. To show true or false:

A. Naffziger test :



- واقف خلف المريض
- واثنى راسه للخلف بدرجة بسيطة
- Observe the eye ball from above keeping the supraciliary ridges as your plane of vision
- In true exophthalmos : eye is seen protruding

B. Frazer's test



- واقف بجانب المريض
- To see of sulcus of orbital margin & covered globe with the patient's eye slight closed.
- In true exophthalmos the sulcus is shallower than normal

C. Ruler test



- واقف بجانب المريض
- A ruler is put to touch the superior & inferior orbital margin
- In true exophthalmos the cornea is touched

II. To determine the degree :

A. Exophthalmometer

B. Ruler to measure distance between lateral orbital margin & apex of cornea (Normally = 15-17m)

How to examine eye signs :

- Stellwag's sign : infrequent blinking (Normally = 5-8 times/min)

- Dalrymple's sign: A rim of sclera between cornea & upper eye lid

- Mobius sign : Lack of convergence on looking to near object due to weakness of Medial recti muscle



- Joffroy's sign : Loss of wrinkling of the forehead when moving the eye upwards with head fixed

- Von Graefe's sign : Upper eye lid lags behind when moving the eye downwards with head fixed (Grave المقابر وكده تحت)

- Rosenbach's sign : Tremors in upper eye lid when eyes are gently closed



- Topolansky's sign: Congestion of to pericorneal region of the eye in patients

- Jellinek's sign: brownish pigmentation of the upper eyelid

- Tellas's sign : brownish pigmentation of the lower eyelid



Discussion on General examination

1. What is meant by sleeping pulse ?
 - It is clinical confirmation of rapid pulse even during sleep so it excludes anxiety
2. Why are vital signs stable in case of toxicity during examination ?
 - Because, the patient under treatment e.g. Indral
3. What is the cause of unequal pulse in case of goitre ?
 - If Retro-sternal extension (R.S.E)
4. What are other causes of water hammer pulse ? see general chapter
5. How can you diagnose under built ?
 - Prominent maxilla & zygoma
 - ↓ Muscle bulk.
 - ↑ Fold of skin at biceps & triceps muscles.
6. Which type of malignancy characterized by bone metastasis ?
 - Follicular carcinoma
7. What is meant by exophthalmos ?
 - Actual protrusion of eye ball.
8. What is the cause of exophthalmos ?

Unknown but may be E.P.S. (Exophthalmos producing substance)
9. What are the causes of unilateral exophthalmos ?
 - Orbital cellulites.
 - Orbital neoplasm.
 - Cavernous sinus thrombosis.
 - Orbital aneurysm i.e. ophthalmic artery aneurysm
 - A-V fistula between ICA & cavernous sinus
 - Neurofibromatosis of optic nerve.
10. What are the causes of pulsating exophthalmos ?
 - Orbital aneurysm & A-V fistula between I.C.A & cavernous sinus
11. What is the difference between true & apparent?
 - True exophthalmos = rim of sclera above & below the cornea
 - False exophthalmos = rim of sclera above the cornea (Upper eye lid retraction)
12. What is DD between fine & flapping tremors ?
 - Fine tremors : Due to ↑ metabolites → irritation of nerve ending → tremors of small joints of hand.
 - Flapping tremors: Due to ↑ Toxins → irritation of extra-pyramidal Δ → tremors of wrist joint of hand
13. What is meant by pre-tibial myxedema ?
 - Pretibial myxoedema presents with a swollen and lumpy appearance over the shins and sometimes also affects the feet. The skin may be discolored pink or purple, with prominent hair follicles. This is known as 'peau d'orange' (orange-peel) appearance. It may instead look warty or 'verrucous'.
 - It is due to deposition of mucin at skin.
 - Associated with clubbing fingers & toes
14. What is meant by mediastinal syndrome ?
 - Dyspnea , congested neck veins , brassy cough
15. What are the causes of liver enlargement ?
 - Thyrotoxic HF.
 - Auto-immune [1ry toxic goitre & Hashimoto's thyroiditis]
 - Thyroid lymphoma.
 - Liver metastasis.
16. What are causes of spleen enlargement ?
 - Auto-immune [1ry toxic goitre & Hashimoto's thyroiditis]
 - Thyroid lymphoma



II. Local examination:

- **Proper position** : patient is sitting down & neck is extended :
- N.B.: Pizzillo's method : if patient obese with short neck, ask him to put his hand behind his neck
- **Proper exposure** : whole head & neck up to supra-clavicular fossa (from the nipple upwards)

I. Inspection (N, 6S, E, 3D): patient is sitting with extended neck from 2 sites: front & side :

1. Number : Single swelling

2. 6 S

• **Site** : Lower part of front of neck i.e. muscular triangle

• **Shape** :

- If diffuse (U shaped or butterfly)
- If localized (irregular or oval)

• **Size** : Variable in (cm x cm)

• **Surface** :

- Smooth if 1ry toxic goitre.
- Nodular if: 2ry toxic goiter or SNG.
- Irregular if malignancy

• **Skin over** :

- Dilated veins if retro-sternal goitre.
- Scars if previous thyroidectomy or Biopsy
- Redness if inflammation.
- Infiltration if malignancy

• **Special signs**

A. **Pulsation** : look tangentially : may be

- Expansile at the upper pole as in 1ry toxic goitre.
- Transmitted if over carotid artery.

B. **Move up & down with deglutition** as goiter

3. **Lower Edge** : (Better seen with deglutition) well defined or not.

• We comment on lower pole seen or not to exclude retro-sternal extension : ill-defined edge

4. **Deep structure**: ask the patient to flex the neck against resistance then look to the size:

• If smaller → Deep to muscle

• If same size → Infiltrate the muscle i.e. malignancy.

5. **Draining L.Ns** "For metastasis" see chapter lymphadenopathy

6. **Distal Effect**:

• **Pemberton's sign in R.S. Extension**: Ask patient to raise up arms & keep this position for a while → congestion of face due to obstruction of great veins & trachea at thoracic inlet

II. Palpation :

A. Palpation from front:

1. **Lahey's method**:

• Push thyroid gland to one side and palpating the lobe of that side with the other hand and then repeat for other lobe

2. **Crile's method**: for small gland with the thumb, placed over the lobe while the patient is swallowing

كلمة السر : ابلغ ريقك



B. Palpation of thyroid gland from behind: Classic method

Examination of thyroid lobes:	Examination of lower border	Examination of the isthmus :
 • Palpation of thyroid gland from behind with flexed neck & slightly tilt the head laterally to the side examined to relax the deep fascia of the neck (investing layer) • Put your thumbs upon the nape & put other fingers over the gland • Press by one hand over one lobe and feel the other lobe by other hand	 • By radial sides of indices & ask the patient to swallow	 • Stand behind the patient • Put one of your hands on the head of the patient • Tilt the head of the patient forward • Put the index of the other hand on the midline of the trachea and palpate • Ask the patient to swallow

❖ Then comment on the following :

1. 3T:

- Temperature حركة بظهر اليد: warm as in toxic goiter
- Tenderness حركة بطن اليد وعيني على وش العيان: tender as in malignancy
- Thrill : at upper part as in toxic goiter

2. 6S:

- Site, Shape, Size, Surface حركة براحة اليد [then comment as inspection]
- Skin over: (attached to skin or not) by :
 - Pinching skin from swelling.
 - Sliding the skin over the swelling.
 - Push swelling under skin: If puckering = infiltrated skin. i.e. malignancy

• Special sign [as inspection]

3. Lower border (Edge) حركة بجانب اليد: Well defined or ill defined

4. Consistency حركة باليدين: may be :

- Hard as in malignancy or calcified SNG
- Firm as in 2ry toxic goitre or SNG.
- Soft in 1ry toxic goitre or physiological goiter

5. Deep structure :

A. Muscle: Sternomastoid: tilt the head to the same side and try to pinch the muscle and ask the patient to swallow

- If you pinch the muscle freely and not moved with swallowing → not fixed

B. Carotid vessels : common carotid pulsation: May be:

- Displaced carotid pulsation as in benign lesions
- Absent carotid pulsation (i.e. Berry's sign) as in malignancy.

C. Trachea :

- Position: central or not.
- Mobility (attached or not to the swelling) : grasp whole swelling to show mobility in side to side on trachea (Rocking) then up & down

6. Draining L.Ns upper & lower deep cervical L.Ns (See chapter lymphadenopathy)

7. Distal effect: Kocher's test:

- Slight compression on lateral lobes produce stridor → It means tracheomalacia

Percussion : Direct percussion on manubrium sterni is normally resonant, If dull = Retro-sternal goiter

Auscultation : A Systolic Murmur may be heard over upper pole i.e. Thyroid bruit as in 1^{ry} toxic goiter

Discussion on local examination

- What is meant by muscular triangle ?
 - It is called "muscular Δ " because it contains:
 - Sterno-hyoid muscle.
 - Sterno-thyroid muscle.
 - Thyro-hyoid muscle.
 - Omo-hyoid muscle
- Why does Goitre moves up & down with deglutition ?
 - Because it is included in pre-tracheal fascia.
 - NB.: Attachment of pre-tracheal fascia:
 - Above : Oblique line of thyroid cartilage & hyoid bone
 - Below: Superior mediastinum,
 - On each side : carotid sheath
- When is Goitre unable to moves up & down with deglutition ?
 - Malignancy
 - Retrosternal extension (R.S.E.)
 - Huge in size.
 - Riedel's thyroiditis (due to fibrosis)
- Which Swellings moves up with protrusion of tongue ?
 - Thyroglossal cyst.
 - Sub-hyoid bursitis.
- What are other Swellings move up & down with deglutition?
 - Goitre.
 - Thyroglossal cyst.
 - Pre-tracheal L.Ns
 - Pre-laryngeal L.Ns.
 - Sub-hyoid bursa.
 - Laryngocele.
- What are the causes of Hard goitre ?
 - Malignancy.
 - Riedel's thyroiditis.
 - Calcified SNG.
 - Tense cyst
- What is the Anatomical site of Carotid artery ?
 - It felt Against carotid tubercle of C6.
- What is the 1st L.Ns felt Clinically in Malignancy ?
 - Pre-laryngeal L.Ns
- What is the value of Kocher's test?
 - The value is preoperative consent (from patient) for tracheostomy
- What are the causes of dullness on Manibrium sterni ?
 - Retrosternal goitre.
 - Ectopic thyroid tissue.
 - Pre-tracheal L.Ns.
- What are the complications of SNG ?
 - Carcinoma "follicular type 3%"
 - 2ry toxic goitre.
 - Hemorrhage on cyst.
 - Calcification.
 - Retrosternal extension

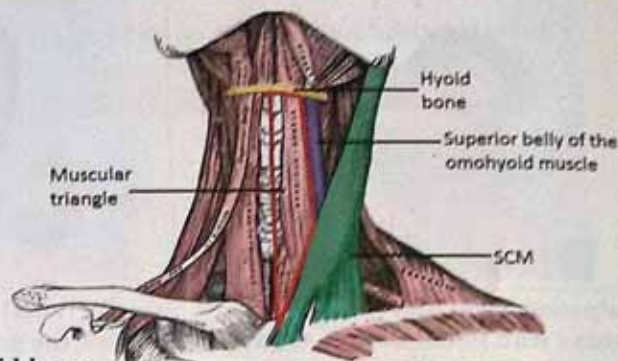


Fig. 1: Photograph of anterior chest wall showing tortuous veins and neck showing goitre.

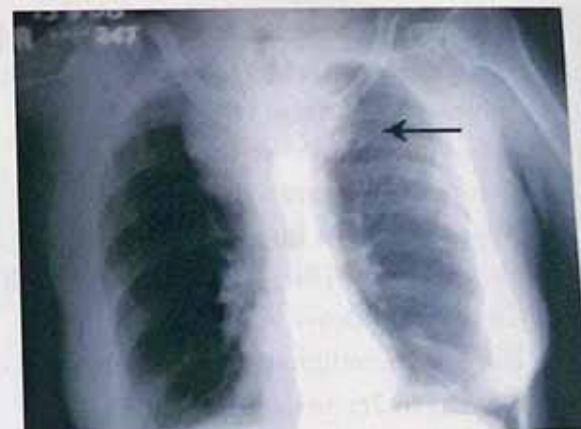
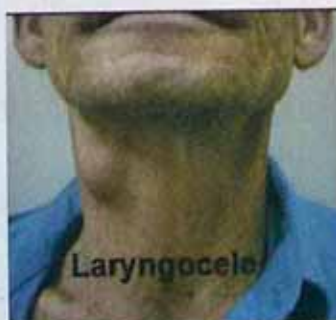
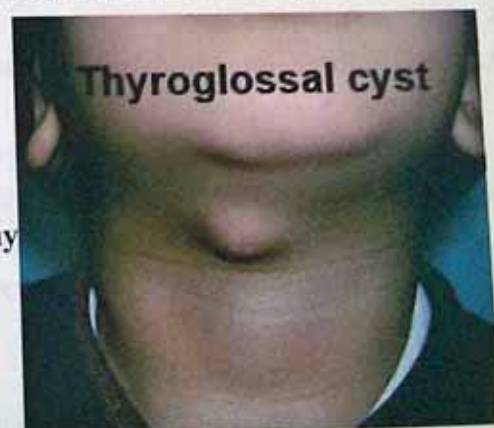


Fig. 2 : Chest x-ray PA view showing retrosternal goitre.



DIAGNOSIS

❖ **During examination : We suspect the following clinical cases**

1. Toxic goitre (1ry or 2ry)
2. Malignancy.
3. Simple nodular goitre (SNG)

❖ **So for diagnosis we must to exclude**

1. **Manifestation of toxicity:** For D.D between 1ry and 2ry :

⊗ 1ry:	⊗ 2ry:
⊗ Grave's (Basedow's) disease	⊗ Plummer's disease
○ Symmetrical ○ Soft & Smooth ○ Young (20 - 40 years) ○ The mass at same time of toxicity ○ Eye signs.	○ Asymmetrical ○ Firm & nodular. ○ Middle (40-50years) ○ The mass before time of toxicity ○ No eye signs (extreme rare)

2. **Manifestation of malignancy**

- Swelling is THIEF (Tender, Hard, Irregular, Enlarged & Fixed)
- Berry's sign (Absent carotid pulsation)
- L.Ns (Enlarged, hard, 1st mobile then fixed)

3. **By Exclusion of 1 & 2: The case is SNG**

Acromegaly

❖ **Personal history**

❖ **Complaint:** Headache, vomiting of 8 years duration.

❖ **HPI:**

- The condition started 8 years ago by gradual onset, progressive course of **headache**, bursting in character, increased in the morning associated with **blurring of vision**. The patient also noticed rapid increase in the sizes of his shoes and rings.
- 3 years later, the patient developed **polyphagia, polyuria, polydypsia** investigated by random blood sugar, diagnosed as **diabetes mellitus**, treated by glucophage. Few months later, the patient developed **generalized bony pain, joint pain and tingling and numbness in both hands and feet**.
- The patient sought medical advice and investigated by skull x-ray and CT, diagnosed as pituitary adenoma and treated by surgery.
- No symptoms suggesting pulmonary or systemic congestion.

❖ **Past history:** No drugs, operation

❖ **Family history :** No consanguinity, No similar condition in family, No common disease in family.

❖ **General examination :**

A. **Vital signs:**

- **Temperature:** 37° c.
- **Bl. Pressure:** 160/90.
- **Pulse:** Regular, 70 beat/minute, average volume, no special character, equal in both sides, intact peripheral pulsation, vessel wall is not felt.

B. **Mentality:** The patient is fully conscious, well oriented for time, place and person. Average mood and memory. The patient is co-operative with average intelligence.

C. **Built:** may be tall stature (specially in gigantism).

D. **Head:**

- Elongated head, Prominent supra-orbital ridges, Enlarged nose, lip, ear, prognathism, separated teeth.
- Skin folds in back of the neck, Husky voice, Defect in visual acuity, field.

E. **Neck :**

↳ **By inspection:**

- Swelling In front of lower neck, butterfly appearance, move up and down with deglutition, disappear on contraction of sternomastoid muscle with no dilated veins or scar.

↳ **By palpation :**

- Thyroid swelling is firm, homogenous, lower border is reached with deglutition, not attached to sternomastoid muscle, with central trachea, intact carotid pulsation, with no thrill or tenderness.

↳ **By percussion:** resonant manubrium sterni.

↳ **By auscultation :** no bruit.

F. **Extremities:**

- UL: spade like (Acromegalic hand), LL: large leg (Acromegalic leg).

G. **Skin:** Thickened, folds, sweaty, greasy skin.

❖ **Systemic examination :**

- **Neuromuscular:** search for myopathy and neuropathy (see Neurology).
- **Bone:** Crepitus in knee joint.
- **Heart:** No signs of hypertension (cardio), No apical gallop, pulsus alternans, basal crepitation (LVF). No signs of systemic congestion (RVF).

❖ **Investigations:** X-ray skull, heel, hand, joint, CT , MRI, GH level, Blood sugar

❖ **Treatment**

- Somatotatin analogue: Octreotide, GH antagonist: pegvisomant, Surgical removal of adenoma.

❖ **Diagnosis :**

- Case of acromegaly for D.D most probably due to pituitary adenoma
 - Adenoma → ↑ I.C.T
 - Acromegaly → hand, head, effects of growth hormone on skin, joint, DM,...

Cushingoid Disease

- ❖ **Personal history**
- ❖ **Complaint:** Progressive weight gain of 3 years duration..
- ❖ **HPI:**
 - The condition started 9 years ago by **skin rash** at face, **loss of hair**, **dyspnea**, **odema of L.Ls** then the patient sought medical advice and investigated and diagnosed as a systemic lupus erythematosus and treated by cortisone.
 - 3 years later, she experienced **headache**, **blurred vision**, **tinnitus** associated with **polyphagia**, **polyuria**, in the form of capozide, glucophage .
 - The patient complained of gradual onset, progressive course of **increased body weight** in last 3 years.
 - The patient also gives history of **generalized bony pain** and **difficulty in lifting heavy objects** and **tingling and numbness** in both hands and feet also increased in the last 3 years of her story.
 - No symptoms suggesting cardiovascular system affection, No menstrual troubles
 - No symptoms suggesting pulmonary T.B , No history of abdominal swellings
- ❖ **Past history:** No drugs, operation
- ❖ **Family history :**
 - No consanguinity, No similar condition in family, No common disease in family.
- ❖ **General examination :**
- A. **Vital signs:**
 - **Temperature:** 37° c.
 - **Bl. Pressure:** 160/90 (with TTT)
 - **Pulse:** Regular, 70 beat/minute, average volume, no special character, equal in both sides, intact peripheral pulsation, vessel wall is not felt.
- B. **Mentality:** The patient is fully conscious, well oriented for time, place and person. Average mood and memory. The patient is co-operative with average intelligence.
- C. **Built:** central obesity
- D. **Head:**
 - Moon face, acne vulgaris, plethoric face, hirsutism, loose teeth, decrease acuity of vision
- E. **Neck :** buffalo hump, supra-clavicular pads of fat
- F. **Skin:** thin, stria rubra, no pigmentation, no diabetic skin changes, no purpura
- ❖ **Systemic examination :**
- **Cardio:** search for signs of HTN and HF (cardiology)
- **Bone:** there is generalized bony tenderness and crepitus with joint movements.
- **Neuromuscular:** search for myopathy, neuropathy (Neurology).
- **Chest :** search for T.B (chest)
- **Abdomen:** no mass in renal angle (adrenal), No masses in abdomen by inspection, palpation (ovarian masses, paramalignant).
- ❖ **Investigations:**
 - CBC: ↑ RBCs, ↓ eosinophil, lymphocyte
 - Blood level of : ↑ Na, ↓ k, ↑ Bl. Glucose.
 - Level of cortisol at morning (8 am) at night (11pm).
 - Level of 17-Hydroxycorticosteroids (OH CS) in urine, free cortisol.
 - Overnight dexamethasone suppression test.
 - U.S, x-ray abdomen.
 - CT, x-ray, MRI skull.
- ❖ **Treatment**
 - Stop exogenous cortisol gradually or try to moderate the dose to the lowest effective dose.
- ❖ **Diagnosis :** A case of obesity + HTN+ DM for D.D most probably Cushingoid disease

Addison's Disease

❖ Personal history

❖ **Complaint:** Change of the color of skin and tongue 20 years duration.

❖ HPI:

- The condition started 30 years ago by **night fever, night sweat, loss of appetite and loss of weight**. Few days later, the patient got an attack of **hemoptysis**, blood was bright red in colour, containing froth, then the patient sought medical advice and investigated by chest x-ray, CBC, and diagnosed as **pulmonary T.B** and took ttt in the form of streptomycin, INH, rifampicin and advised to follow this TTT for one year but the patient discontinued the treatment.
- 10 years later the patient developed excessive **pigmentation** in hands, face, neck, axillae, tongue and gums associated with marked **fatigue, weight loss, vomiting, polyuria, diarrhea and back pain**. The patient was admitted to hospitals and treated many times with IV fluids and vitamins.
- In the last 5 years, the patient complained of **unsteadiness on sudden standing** with **headache, abdominal pain** the patient was admitted to hospital and investigated and took replacement therapy in the form of glucocorticoids and mineralocorticoids.

❖ **Past history:** No drugs, operation, diseases (HTN, DM)

❖ Family history :

- No consanguinity, No similar condition in family, No common disease in family.

❖ General examination :

❖ Vital signs:

- **Temperature:** 37° c. → improved.
- **BL Pressure:** 100/70 (Recombinant position), 60/40 (Standing position). → now improved.
- **Pulse:** Regular, 70 beat/minute, average volume, no special character, equal in both sides, intact peripheral pulsation, vessel wall is not felt, no radio-femoral delay.
- ❖ **Built:** Average built (now improved → was under built).
- ❖ **Mentality:** The patient is fully conscious, well oriented for time, place and person. Average mood and memory. The patient is co-operative with average intelligence
- ❖ **Color:** there is tanning in face, neck, dorsum of hands, palmar crease, elbow, knee, umbilicus, areola, knuckles, in axillae and in between thighs. There is also slate like pigmentation in m.m of inner lip, buccal mucosa, palate, gum, tongue,

❖ **Systemic examination :** There is tenderness in the renal angle.

❖ Investigations:

A. Blood sample:

- ↓ cortisol
- ↓ Na, ↑ k
- ↓ blood glucose
- Anemia, lymphocytosis, eosinophilia

B. Urine sample:

- ↓ cortisol, 17 OHCs, 17 KCs
- ↑ Na, ↓ k
- ↑ volume.

C. X-ray, CT abdomen, chest.

❖ **Treatment:** Replacement therapy: Corticosteroid & Mineralocorticoid.

❖ **Diagnosis :** A case of hyperpigmentation most probably Addison's under replacement therapy most probably due to T.B in adrenal glands

❖ **Why addison's?** (to be differentiated from renal failure which give the same picture but with no pigmentation in MM and no hypotension)

↳ **Pigmentation in mm.**

↳ **Postural hypotension.** (Bl. Pr. not exceed 110)

Myxedema

- ❖ **Personal history**
- ❖ **Complaint:** Puffiness of upper eye lids of 7 years duration.
- ❖ **HPI:**
 - The condition started 7 years duration by gradual onset, progressive course of **puffiness** of upper eyelid followed by **generalized body swelling** and diagnosed as nephrotic disease and take medication in the form of corticosteroid.
 - One year later, the patient experienced **marked weight gain despite loss of appetite, cold intolerance, marked numbness** in both hands and feet, **difficulty in lifting heavy objects**, and the patient become **apathetic, dull**. The patient also suffered from **headache and blurring of vision** and was discovered as hypertensive patient and also noticed **abnormal increase in menstrual blood and marked irregularity** in previously normal cycles.
 - The patient sought medical advice and investigated by thyroid function tests and diagnosed as myxedema and treated by thyroxin.
 - 3 years after TTT, the patient developed **retrosternal chest pain** radiated to the left shoulder, increased with exercise, investigated by ECG and ECHO and diagnosed as ischemic heart disease and treated by nitrates. This was followed by **polyphagia, polydipsia** investigated by random blood sugar and diagnosed as diabetes mellitus .
- ❖ **Past history:** No drugs, operation
- ❖ **Family history :**
 - No consanguinity, No similar condition in family, No common disease in family.
- ❖ **General examination :**
- ❖ **Vital signs:**
 - **Temperature:** 35° c.(now improved)
 - **Bl. Pressure:** 160/90.
 - **Pulse:** Regular, 60 beat/minute, average volume, no special character, equal in both side, intact peripheral pulsation, vessel wall is not felt. (was bradycardia)
 - Cold intolerance.
- ❖ **Built:** Over-weight.
- ❖ **Color:** Malar Flush, pallor, carotinemia (may be)
- ❖ **Mentality:** The patient is fully conscious, well oriented for time, place and person. Average mood and memory. The patient is co-operative with average intelligence.
- ❖ **Head:**
 - Puffy face.
 - Dry brittle hair then Alopecia.
 - Loss of outer % of eye brows, periorbital swelling.
 - Husky voice.
 - Malar flush., No macroglossia.
- ❖ **Neck :** No scar, no dilated veins, no thyroid swelling.
- ❖ **Skin:** Cold, pale, thick, edematous.
- ❖ **Extremities:** UL & LL: non pitting edema.
- ❖ **Systemic examination :**
 - **Muscle:** myopathy, neuropathy → notice delayed relaxation of tendon jerks (see Neuro)
 - **Cardio:** search for HTN, IHD, pericardial effusion (cardio)
 - **Chest:** search for pleural effusion.
 - **Abdomen :** abdominal distension
- ❖ **Investigations:** Thyroid function tests.
- ❖ **Treatment** Replacement by eltroxin gradually to avoid coronary heart disease.
- ❖ **Diagnosis :** A case of myxedema most probably auto immune.
- ❖ **Why myxedema??**
 - Deposition of myxedematous substance. Exclude iatrogenic cause.
- ❖ **Why autoimmune?**
 - Exclusion of iatrogenic causes, associated DM.

Thyrotoxicosis

❖ **Personal history**

❖ **Complaint:** swelling in the neck 5 years duration.

❖ **HPI:**

❖ The condition started of 5 years ago by **swelling** in front of lower neck of gradual onset, progressive course .Then the patient experienced **palpitation, decrease in body weight** in spite of excellent appetite, **irritability, insomnia, heat intolerance**. Then the patient sought medical advice, admitted to hospital and investigated by ECG, urine, stool analysis, blood sugar.

❖ The patient also gives no history of **diarrhea**.

❖ No dyspnea, dysphagia or change of voice, no pain in neck .

❖ No symptoms suggesting cardiovascular system affection.

❖ No symptoms of muscle diseases.

❖ **Past history:** No drugs, operation

❖ **Family history :**

○ No consanguinity, No similar condition in family, No common disease in family.

❖ **General examination :**

• **Temperature:** 37° (normal)

• **BL. Pressure:** 160/80.

• **Pulse:** Regular(may be irregular), 110 beat/minute, big pulse volume, equal in both sides, with water hammer pulse, intact peripheral pulsation, vessel wall is not felt, no radio-femoral delay.

• Heat intolerance.

• **Built:** Average(may be under built)

→ **Head:** : the patient has certain eye signs:

○ Infrequent blinking "Stellwag's sign".

○ Rim of sclera between cornea, eye lid "Dalrymple sign".

○ Loss of corrugation on upward movement "Joffroy's sign".

○ Lid lag "Von graffe's sign".

○ Lack of sustained convergence "Mobius sign".

○ Exophthalmos confirmed by Naffziger method, ruler method, Frazer's method.

→ **Neck :**

↳ **By inspection:**

• Swelling in front of lower neck, butterfly appearance, move up and down with deglutition, disappear on contraction of sternomastoid muscle with no dilated veins or scar

↳ **By palpation :**

• Swelling is firm, homogenous, lower border reached during deglutition, not attached to sternomastoid muscle, with central trachea, intact carotid pulsation, with thrill, no tenderness.

↳ **By percussion:** resonant manubrium sterni

↳ **By auscultation :** bruit near upper pole of the swelling.

→ **Extremities:**

• UL: fine tremors in out stretched hands, warm, sweaty.

• LL: non pitting edema, hyperreflexia (may be pitting oedema if complicated by HF).

→ **Skin:** warm, moist, sweaty, acropachy, pretibial myxedema.

❖ **Systemic examination :**

○ **CVS:** search for HF, and hemic murmurs.

○ **Neuro:** Exaggerated deep reflexes, tremors

❖ **Investigations: Thyroid function tests.**

❖ **Diagnosis :** A case of mass in muscular triangle most probably thyroid swelling; hyperthyroidism for D.D most probably graves' disease.